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RESPIRATORY CARE

A MONTHLY SCIENCE JOURNAL
40TH YEAR—ESTABLISHED 1956

- Work Redesign: Out with the Bath Water (Maybe) but Do Save the Baby!–Editorial
- Inhaled NO Delivery Systems–Editorial
- Continuous NO for Infant Ventilation
- Patient-Determined Flow during Mechanical Ventilation
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- Mechanical Ventilation in Duchenne's Disease
- Hypoxic Hypercarbic Gas Mixtures in Hypoplastic Left-Heart Syndrome
- 3 New Clinical Practice Guidelines
  - Defibrillation during Resuscitation
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FACE TO FACE WITH CHANGE

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Editorials, Commentaries, & Reviews To Note

Persistent Pulmonary Hypertension of the Newborn (State-of-the-Art Paper)—FC Morin III, KR Stenmark. Am J Respir Crit Care Med 1995;151:2010-2032.

Nitrogen Dioxide Production during Mechanical Ventilation with Nitric Oxide in Adults: Effects of Ventilator Internal Volume, Air Versus Nitrogen Dilution, Minute Ventilation, and Inspired Oxygen Fraction—M Nishimura, D Hess, RM Kacmarek, R Ritz, WE Hurford. Anesthesiology 1995;82(5):1246-1254.

BACKGROUND: Inhaled nitric oxide (NO) may be useful in the treatment of adult respiratory distress syndrome and other diseases characterized by pulmonary hypertension and hypoxemia. NO is rapidly converted to nitrogen dioxide (NO_2) in oxygen (O_2) environments. We hypothesized that in patients whose lungs are mechanically ventilated and in those with a long residence time for NO in the lungs, a clinically important [NO_2] may be present. We therefore determined the rate constants for NO conversion in adult mechanical ventilators and in a test lung simulating prolonged intrapulmonary residence of NO. METHODS: NO (800 ppm) was blended with nitrogen (N_2), delivered to the high-pressure air inlet of a Puritan-Bennett 7200ae or Siemens Servo 900C ventilator, and used to ventilate a test lung. The ventilator settings were varied: minute ventilation ($\dot{V}_E$) from 5 to 25 L/min, inspired O_2 fraction (F_{IO_2}) from 0.24 to 0.87, and [NO] from 10 to 80 ppm. The experiment was then repeated with air instead of N_2 as the dilution gas. The effect of pulmonary residence time on NO_2 production was examined at test lung volumes of 0.5-4.0 L, $\dot{V}_E$ of 5-25 L/min, F_{IO_2}, of 0.24-0.87, and [NO] of 10-80 ppm. The inspiratory gas mixture was sampled 20 cm from the Y-piece and from within the test lung. NO and NO_2 were measured by chemiluminescence. The rate constant (k) for the conversion of NO to NO_2 was determined from the relation $1/[NO]_t - 1/[NO]_0 = k \times [O_2] \times t$, where t = residence time. RESULTS: No NO_2 was detected during any trial with $\dot{V}_E$ 20 or 25 L/min. With N_2 dilution and the Puritan-Bennett 7200ae, NO_2 (≤ 1 ppm) was detected only at a $\dot{V}_E$ of 5 L/min with an F_{IO_2} of 0.87 and [NO] $\geq$ 70 ppm. In contrast, [NO_2] values were greater with the Servo 900C ventilator than with the Puritan-Bennett 7200ae at similar settings. When NO was diluted with air, clinically important [NO_2] values were measured with both ventilators at high [NO] and F_{IO_2}. Rate constants were 1.46×10^{-9} ppm$^{-2} \times$ min^{-1} when NO was mixed with N_2, 1.17×10^{-8} ppm$^{-2} \times$min^{-1} when NO was blended with air, and 1.44×10^{-9} ppm^{-2} min^{-1} in the test lung. CONCLUSIONS: [NO_2] increased with increased F_{IO_2}, and [NO], decreased $\dot{V}_E$, blending with air, and increased lung volumes. Higher [NO_2] was produced with the Servo 900C ventilator than the Puritan-Bennett 7200ae because of the greater residence time. With long intrapulmonary residence times for NO, there is a potential for NO_2 production within the lungs. The rate constants determined can be used to estimate [NO_2] in adult mechanical ventilation systems.

Passive Smoke Exposure and Otitis Media in the First Year of Life—JL Ey, CJ Holberg, MB Aldous, AL Wright, FD Martinez, LM Taussig, Group Health Medical Associates. Pediatrics 1995;95:670-677.

OBJECTIVE: This study examined, in a health maintenance organization population of children, the associations between parents' smoking and otitis media (OM) in their children while controlling for other known risk factors. METHODS: Healthy newborns (1,246) in a large health maintenance organization were enrolled at birth, and 1,013 (81%) were followed prospectively for the first year of life. Their medical records were reviewed for the diagnosis of otitis media. Information on risk factors for recurrent OM (ROM) was collected, including a number of variables related to parental smoking. RESULTS: After controlling for other known risk factors for ROM including gender, day care, other siblings in the home, parental history of hay fever, and method of feeding, it was found that heavy maternal smoking of 20 or more cigarettes per day was a significant risk factor for ROM but not for nonrecurrent otitis. Heavy maternal smoking was associated with a threefold risk for ROM if the infant weighed less than the mean at birth (3.5 kg) after controlling for other risk factors. No association was found with paternal smoking. CONCLUSIONS: Heavy maternal smoking is a significant risk factor for ROM in the first year of life. This smoking effect seems to be stronger among infants of lower birthweight.

Perinatal Outcomes in the Pregnancies of Asthmatic Women: A Prospective Controlled Analysis—M Schatz, RS Zeiger, CP Hoffman, Khardeh, A Forsythe, L Chilingar, et al. Am J Respir Crit Care Med 1995;151:1170-1174.

Prior studies have found an increased incidence of adverse perinatal outcomes of pregnancies in asthmatic mothers, but these studies have been poorly controlled for asthma therapy and other confounding factors. The purpose of this study was to assess perinatal outcomes in actively managed pregnant asthmatic women as compared with matched nonasthmatic controls. Using an inception cohort design, we studied a volunteer sample of 486 pregnant (< 28 wk) women with documented asthma and 486 pregnant nonasthmatic controls with normal pulmonary function. Cases and controls were matched for age, smoking status, parity, and year of delivery. Asthma was managed with step therapy to prevent acute asthmatic episodes and asthma symptoms that interfered with sleep or normal activity. Chronic hypertension was significantly more common ($p = 0.007$) in asthmatic subjects (3.7%) than in matched controls (1.0%). However, no significant differences in incidences of preeclampsia, perinatal mortality, preterm births, low-birth-weight infants, intrauterine growth retardation, or congenital malformations were observed in the pregnancies of the asthmatic women as compared with the matched controls. Trends were observed toward relationships between more severe asthma requiring emergency therapy or corticosteroids and increased incidences of preeclampsia and low-birthweight infants, but these associations were not statistically significant. These data suggest that the overall perinatal prognosis for women with actively managed asthma during pregnancy is comparable to that for the nonasthmatic population.

Effect of Mouthpiece Breathing on Cardiorespiratory Response to Intense Exercise—KE Bloch, J Barandun, MA Sackner. Am J Respir Crit Care Med 1995;151:1087-1092.

Use of mouthpiece and noseclips alters breathing pattern at rest and during moderate exercise.

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REFERENCES: 1. Data on file. HealthScan Products Inc. 2. Wheeler BB, Bollers ND: Initial experience with a novel spacer device. (Presented at the 1993 meeting of the American Academy of Allergy and Immunology) * U.S. Patent No. 5,040,527 OA760004-0 © 1994, HealthScan Products Inc

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Our purpose was to extend observations on mouthpiece breathing to its effects on the cardiorespiratory response to intense exercise and to develop and validate an algorithm for computer-assisted analysis of breathing pattern recorded with respiratory inductive plethysmography. Six normal men performed incremental bicycle exercise to volitional exhaustion on two occasions with and without mouthpiece and noseclip. ECG and breathing pattern recorded with a respiratory inductive plethysmograph were analyzed manually and with computer assistance. Mouthpiece breathing increased tidal volume and respiratory cycle time by up to 63 and 33%, respectively ($p < 0.02$), during mild exercise, but it did not alter performance, heart rate, or breathing pattern at maximal exercise. Mean differences between inductive plethysmographic and spirometric tidal volumes were < 5% during validation at rest and maximal exercise. Application of the proposed algorithm for semiautomatic breathing pattern analysis provided significant time savings with no loss in precision compared with manual analysis. In conclusion, in normal men, performance and breathing pattern during maximal exercise are not altered by a mouthpiece and noseclip, and accurate computer-assisted measurement of ventilation from external transducers may be performed, even during intense exercise.

Australian Collaborative Trial of Antenatal Thyrotropin-Releasing Hormone (ACTOBAT) for Prevention of Neonatal Respiratory Disease—ACTOBAT Study Group. Lancet 1995; 345:877-882.

The addition of thyrotropin-releasing hormone (TRH) to antenatal glucocorticoid treatment of women at risk of preterm delivery has been reported to lower the risk of respiratory distress syndrome (RDS) in the infant. We have assessed the efficacy of 200 μg TRH in a multicentre randomized trial. 1,234 women at 24 weeks to 31 weeks 6 days of gestation with a singleton or twin pregnancy and at risk of preterm delivery were randomly allocated to groups receiving 200 μg TRH or placebo intravenously every 12 h up to a maximum of 4 doses. Randomization was stratified by duration of gestation and centre. All women received glucocorticoids. Clinical outcome is known for 1,231 women and their 1,397 infants. The frequencies of the main prespecified study outcomes—RDS (relative risk 1.17 [95% CI 1.00-1.36], $p = 0.05$) and need for ventilation (1.15 [1.01-1.31], $p = 0.04$)—were higher in TRH-group infants than in control infants. The excess risk in the TRH group was greater in infants who were born more than 10 days after treatment. Multivariate analysis adjusting for duration of gestation at randomization, time from randomization to delivery, parity, history of perinatal death, and infant's sex did not affect the risk estimates. TRH administration was associated with increased risks of maternal nausea, vomiting, lightheadedness, and a rise in blood pressure to 140/90 mm Hg or higher. Antenatal TRH given with glucocorticoids to women at high risk of preterm delivery is associated with maternal and perinatal risks and cannot be recommended for widespread clinical use. *See related commentary: **Antenatal TRH**—M Chiswick. Lancet 1995;345:872.*

Passive Smoking and Heart Disease: Mechanisms and Risk—SA Glantz, WW Parmley. JAMA 1995;273:1047-1053.

OBJECTIVE: Recent clinical, laboratory, and epidemiological evidence that passive smoking causes heart disease was reviewed, with particular emphasis on understanding the underlying physiological and biochemical mechanisms. DATA SOURCES: Publications in the peer-reviewed literature were located via MEDLINE, citation in other relevant articles, and appropriate reports by scientific agencies. Greatest emphasis was given to work published since 1990. CONCLUSIONS: Passive smoking reduces the blood's ability to deliver oxygen to the heart and compromises the myocardium's ability to use oxygen to create adenosine triphosphate. These effects are manifest as reduced exercise capability in people breathing secondhand smoke. Secondhand smoke increases platelet activity, accelerates atherosclerotic lesions, and increases tissue damage following ischemia or myocardial infarction. The effects of secondhand tobacco smoke on the cardiovascular system are not caused by a single component of the smoke, but rather are caused by the effects of many elements, including carbon monoxide, nicotine, polycyclic aromatic hydrocarbons, and other, not fully specified elements in the smoke. Nonsmokers exposed to secondhand smoke in everyday life exhibit an increased risk of both fatal and nonfatal cardiac events.

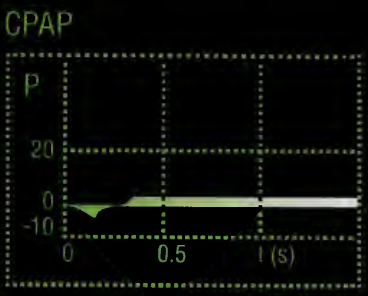
Preservation of Pulmonary Function in the Ventilated Neonatal Piglet with Normal Lungs—D Easa, KC Finn, V Balaraman, S Sood, S Wilkerson, W Takenaka, TG Mundie. Pediatr Pulmonol 1995;19:174-181.

Little attention has been focused on the progressive pulmonary deterioration which occurs in mechanically ventilated infants with normal or mildly abnormal lungs. We hypothesized that lung function would deteriorate over a 24-hr period in anesthetized neonatal piglets with normal lungs mechanically ventilated at 2 cm H_2O PEEP (2PEEP group). We further hypothesized that an intermittent lung inflation procedure consisting of 15 out of 60 min of increasing lung distention (4, 8, 12 cm H_2O PEEP), with the remaining 45 min at 2 cm H_2O PEEP (Inflation group) would prevent this deterioration in lung function, similar to piglets mechanically ventilated continuously at 6 cm H_2O PEEP (6PEEP). Results indicate that 2PEEP piglets experienced progressive deterioration in lung function, including dynamic lung compliance (–42%) and lung resistance (+55%). In contrast, Inflation piglets and 6PEEP piglets had no deterioration in lung function. Hemodynamics were similar between groups, although they were the most stable in the 6PEEP group. Histopathological changes were not significantly different. We conclude that (1) prolonged mechanical ventilation at 2 cm H_2O PEEP in neonatal piglets resulted in progressive deterioration in pulmonary function, (2) intermittent lung inflation or continuous 6 cm H_2O PEEP prevented deterioration, and (3) functional changes occurred without changes in histopathology. Lung inflation strategies other than PEEP can be used to prevent deterioration in lung function which accompanies prolonged mechanical ventilation in anesthetized nonspontaneously breathing piglets with normal lungs.

Validation of the Phase Angle Technique as an Objective Measure of Upper Airway Obstruction—J Hammer, CJL Newth, TW Deakers. Pediatri Pulmonol 1995;19:167-173.

Thoraco-abdominal asynchrony (TAA) during upper airway obstruction (UAO) in small children can be documented by phase angle analysis of the Lissajous figure from the output of a noncalibrated respiratory inductance plethysmograph. Phase angle measurements have not been related to levels of inspiratory resistance, nor to the effect of breathing a 79% helium-21% oxygen mixture (heliox) during inspiratory resistance. We examined the effects of graded inspiratory loading (5-1,000 cm $H_2O \cdot s \cdot L^{-1}$) on TAA as measured by phase angle in 10 male, anesthetized, and intubated Rhesus monkeys, breathing room air and heliox. Phase angles increased with inspiratory loading from a baseline value of 22 ± 3° to 165 ± 8° at 1,000 cm $H_2O \cdot s \cdot L^{-1}$ resistance and correlated significantly with the level of inspiratory loading ($r = 0.82$). End-tidal carbon dioxide P_{ETCO_2} increased from 39 ± 1 to 49 ± 3 mm Hg at the highest load, but correlated only weakly with phase angle measurements ($r = 0.60$) and the level of inspiratory loading ($r = 0.56$). By changing to heliox breathing at the highest tolerated resistance, P_{ETCO_2} dropped significantly from 49 ± 3 to 40.5 ± 4 mm Hg ($P < 0.001$) with no significant change in phase angles: 169 ± 13° and 165 ± 8°, respectively ($P > 0.05$). We conclude that heliox therapy for acute alveolar hypoventilation during UAO improves ventilation, but does not decrease TAA at high inspiratory resistance. Continuous monitoring of the relative changes in phase angles is useful to observe the severity of UAO in the early stages. However, phase angle analysis does not predict the development of hypercapnia secondary to inspiratory load.

Predictors for Failure of *Pneumocystis carinii* Pneumonia Prophylaxis—AJ Saah, DR Hoover, Y Peng, JP Phair, B Visscher, LA Kingsley, LK Schrager, for the Multicenter AIDS Cohort Study. JAMA 1995;273:1197-1202.



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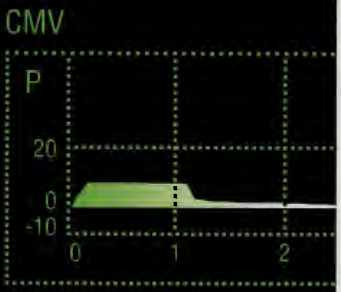

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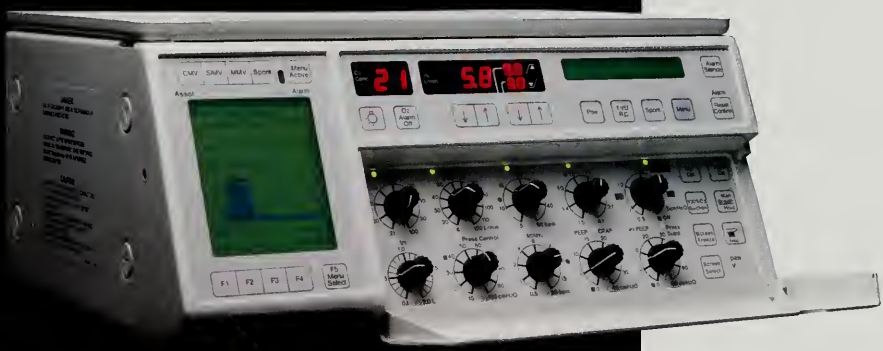

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OBJECTIVE: To identify clinical and epidemiological factors associated with failure of *Pneumocystis carinii* pneumonia (PCP) prophylaxis in those receiving primary and secondary prophylaxis. DESIGN: Longitudinal cohort study of participants infected with human immunodeficiency virus type 1 in the Multicenter AIDS Cohort Study who used PCP prophylaxis regimens after their T-helper lymphocyte counts had decreased to less than 0.200×10^9/L (200/μL). MAIN OUTCOME MEASURE: Occurrence or recurrence of PCP. RESULTS: A total of 476 participants reported taking one or more of the following regimens: trimethoprim-sulfamethoxazole (TMP-SMX), dapsone, and/or aerosolized pentamidine—367 as primary prophylaxis and 109 as secondary prophylaxis after a previous episode of PCP. A total of 92 (20%) developed PCP despite prophylaxis. The mean failure rates per person-year of follow-up were 16.0% for those receiving primary prophylaxis and 12.1% for those receiving secondary prophylaxis ($P = 0.19$). Median times to death after initiation of primary or secondary prophylaxis were 2.0 and 1.2 years, respectively. The main predictor for failure of PCP prophylaxis was profound T-helper lymphocytopenia; 86% of failures occurred after T-helper cell counts decreased to less than 0.075×10^9/L and 76% occurred after counts decreased to less than 0.050×10^9/L. In multivariate time-dependent analysis, when compared with counts between 0.100×10^9/L and 0.200×10^9/L, the risk ratio for failure with counts less than 0.050×10^9/L was 2.90 ($P < 0.001$). Once T-helper cell counts were considered, fever was the only other health status indicator that predicted subsequent PCP (ie, a time-dependent risk ratio of 2.22; $P = 0.01$). Use of TMP-SMX as the prophylaxis regimen was protective but did not eliminate failure (ie, a time-dependent risk ratio of 0.55; $P = 0.03$). CONCLUSIONS: These findings strongly support identifying improved methods of PCP prophylaxis once T-helper cell counts decrease to less than 0.075×10^9/L or 0.100×10^9/L. Given this severe degree of immunosuppression, an inherently more effective regimen against *P carinii* is required.

Interfacing between Spontaneous Breathing and Mechanical Ventilation Affects Ventilation-Perfusion Distributions in Experimental Bronchoconstriction—C Putensen, J Räsänen, FA López. Am J Respir Crit Care Med 1995; 151:993-999.

The effect of interfacing between spontaneous and mechanical ventilation on ventilation-perfusion ($\dot{V}_A/\dot{Q}$) distributions was determined during pressure-support ventilation (PSV) and in the presence and absence of spontaneous breathing during biphasic positive airway pressure (BIPAP®) in 10 pigs with methacholine-induced bronchoconstriction. Whereas BIPAP without spontaneous breathing provides full and PSV breath-to-breath synchronized ventilatory support, BIPAP allows unrestricted spontaneous breathing throughout the mechanical cycle. Compared with BIPAP with and without spontaneous breathing, PSV effected an increase in ventilatory rate ($p < 0.05$) and a higher minute ventilation ($\dot{V}_E$) ($p < 0.05$). Spontaneous breathing during BIPAP accounted for $15 \pm 1\%$ of the $\dot{V}_E$ and increased cardiac output (C.O.) from 4.5 ± 0.2 to $5.3 + 0.2$ L/min ($p < 0.05$), P_{aO_2} from 55 ± 3 to 80 ± 4 mm Hg ($p < 0.05$), and oxygen delivery ($\dot{D}_{O_2}$) from 442 ± 39 to 630 ± 43 mL/min ($p < 0.05$). PSV did not increase C.O., P_{aO_2}, and $\dot{D}_{O_2}$. Spontaneous breathing did not affect oxygen consumption. During BIPAP, spontaneous breathing accounted for a $15 \pm 2\%$ decrease ($p < 0.05$) in blood flow to shunt units and a $16 \pm 2\%$ increase ($p < 0.05$) in the perfusion of normal $\dot{V}_A/\dot{Q}$ units. Perfusion of shunt and normal $\dot{V}_A/\dot{Q}$ units was similar during PSV and BIPAP without spontaneous breathing. Dead space ventilation decreased with spontaneous breathing during BIPAP by 12% compared with PSV ($p < 0.05$). Dispersion of ventilation distribution was lowest during BIPAP. Uncoupling of spontaneous and mechanical ventilation during BIPAP improved gas exchange by allowing better $\dot{V}_A/\dot{Q}$ matching during experimental bronchoconstriction. Apparently, the spontaneous respiratory effort during PSV is not sufficient to counteract the $\dot{V}_A/\dot{Q}$ maldistribution caused by airway occlusion and positive pressure lung inflation.

Screening for Active Tuberculosis in HIV Testing Centre—MA Espinal, AL Reingold, E Koenig, M Lavandera, S Sanchez. Lancet 1995; 345:890-893.

In industrialized nations HIV-seropositive individuals can be offered skin testing for tuberculosis (TB) and isoniazid prophylaxis, but this approach is neither practicable nor affordable in most developing countries. In Santo Domingo, Dominican Republic, we offered skin testing and a brief clinical examination for active TB to people requesting HIV testing at one centre. 200 newly detected HIV-positive individuals and 200 age- and sex-matched HIV-negative ones were compared. 39 (9.7%) of the 400 individuals seeking HIV testing had active TB; 29 were HIV positive and 10 were HIV negative (adjusted odds ratio 3.3, 95% CI 1.3-8.7; $p = 0.01$). In multivariate analysis, the strongest independent predictors of active TB were 10 mm or more of induration on skin testing, a history of chronic cough, lymphadenopathy, and HIV infection. Of the patients diagnosed with TB, 85% had one or more symptoms readily ascertainable in a brief screening questionnaire. Screening for TB at HIV-testing sites could be an effective approach to early detection of active TB among not just HIV-positive but also HIV-negative people. Integrating screening for TB into HIV testing schemes could help to reduce the spread of TB and allow patients with TB to be diagnosed and treated earlier. *See related commentary:* ***Screening for Tuberculosis and HIV in Resource-Poor Countries***—*KM DeCook. Lancet 1995;345:873-874.*

The Epidemiology of Tuberculosis among Foreign-Born Persons in the United States, 1986 to 1993—MT McKenna, E McCray, I Onorato. N Engl J Med 1995;332:1071-1076.

BACKGROUND: One third of the world's population is infected with *Mycobacterium tuberculosis,* and in the developed countries immigration is a major force that sustains the incidence of tuberculosis. We studied the effects of immigration on the epidemiology of tuberculosis and its recent resurgence in the United States. METHODS: We analyzed data from the national tuberculosis reporting system of the Centers for Disease Control and Prevention. Since 1986 reports of tuberculosis have included the patient's country of origin. Population estimates for foreign-born persons were derived from special samples from the 1980 and 1990 censuses. RESULTS: The proportion of persons reported to have tuberculosis who were foreign-born increased from 21.6% (4,925 cases) in 1986 to 29.6% (7,346 cases) in 1993. For the entire 8-year period, most foreign-born patients with tuberculosis were from Latin America (43.9%; 21,115 cases) and Southeast Asia (34.6%; 16,643 cases). Among foreign-born persons, the incidence rate was almost quadruple the rate for native residents of the United States (30.6 versus 8.1 per 100,000 person-years), and 55% of immigrants with tuberculosis had the condition diagnosed in their first 5 years in the United States. CONCLUSIONS: Immigration has had an increasingly important effect on the epidemiology of tuberculosis in the United States. It will be difficult to eliminate tuberculosis without better efforts to prevent and control it among immigrants and greater efforts to control it in the countries from which they come. *See the related editorial:* ***Immigrants and Tuberculosis Control***—*D Moradpour, JR Wands. N Engl J Med 1995;332(16):1092-1093. Also see the related commentary:* ***Denial of Care to Illegal Immigrants***—*TA Ziv, B Lo. N Engl J Med 1995;332 (16):1095-1098.*

Bronchodilator Delivery by Metered-Dose Inhaler in Ventilator-Supported Patients—R Dhand, A Jubran, MJ Tobin. Am J Respir Crit Care Med 1995;151:1827-1833.

The optimal dose and technique for administration of bronchodilators with a metered-dose inhaler (MDI) in mechanically ventilated patients have not been established. We studied the efficacy and safety of 10 puffs (90 μg/puff) of albuterol administered by an MDI in seven mechanically ventilated patients with chronic obstructive pulmonary disease (COPD). Rapid airway occlusions at constant flow inflation were performed before and at 5-min intervals after administration of albuterol for 60 min. Significant decreases in maximum (R_{rs}max; $p < 0.01$) and minimum inspiratory resistance (R_{rs}min; $p < 0.01$) were present at 5 min and persisted for 60 min after administration of albuterol ($p < 0.01$ for both parameters).

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$R_s$max indicates maximal inspiratory resistance while $R_s$min represents the ohmic flow resistance. Intrinsic positive end-expiratory pressure decreased significantly ($p < 0.05$) 15 min after albuterol administration. Heart rate, blood pressure, and arterial oxygenation did not show significant change after albuterol. In summary, 10 puffs of albuterol given by an MDI and spacer produced significant bronchodilation in ventilator-supported patients with COPD, without producing side effects. In conclusion, higher doses of albuterol given by an MDI and spacer could be used routinely in mechanically ventilated patients with COPD.

A Randomized Clinical Trial of Active Compression-Decompression CPR vs Standard CPR in Out-of-Hospital Cardiac Arrest in Two Cities—TM Schwab, ML Callaham, CD Madsen, TA Utecht. JAMA 1995;273(16):1261-1268.

OBJECTIVE: To compare the effectiveness of active compression-decompression (ACD) cardiopulmonary resuscitation (CPR) with a handheld suction device vs standard manual CPR in victims of out-of-hospital cardiopulmonary arrest. DESIGN: Prospective randomized clinical trial with crossover group design. SETTING: Emergency medical services (EMS) of a large (San Francisco) and medium-sized (Fresno) city in California. PATIENTS: All normothermic adult victims of out-of-hospital, nontraumatic cardiac arrest on whom CPR was performed by first responders. INTERVENTION: Patients were randomized to receive either standard manual CPR according to American Heart Association guidelines or ACD CPR, on first-responder contact. MAIN OUTCOME MEASURES: Return of spontaneous circulation, admission to the intensive care unit, survival to hospital discharge, and neurological function at hospital discharge. RESULTS: The ACD group (n = 117 in Fresno; n = 297 in San Francisco) and standard group (n = 136 in Fresno; n = 310 in San Francisco) were similar with regard to demographic and prognostic variables, such as age, witnessed arrest and bystander CPR frequency, and initial cardiac rhythm. Average interval from 911 call activation to EMS responder arrival was 6.4 minutes in Fresno and 4.0 minutes in San Francisco. In Fresno, there was no difference between the ACD group and standard CPR group in return of spontaneous circulation (17% vs 20%; P = 0.68), hospital admission (16% vs 20%; P = 0.56), hospital discharge (5% vs 7%; P = 0.64), or cerebral performance category score at discharge (1.5 vs 1.6; P = 0.90). Similarly, in San Francisco there was no difference between the ACD group and standard CPR group in return of spontaneous circulation (19% vs 21%; P = 0.65), hospital admission (13.5% vs 14.5%; P = 0.79), hospital discharge (4.7% vs 5.5%; P = 0.80), or cerebral performance category score at discharge (2.2 vs 2.6; P = 0.31). There was no increase in significant complications associated with the use of ACD CPR. CONCLUSION: There was no improvement in outcome with ACD CPR in out-of-hospital cardiac arrest in these two cities. Differences in study design, demographics, EMS systems, response intervals, training, and technique performance may contribute to the lack of improvement in initial resuscitation with ACD CPR compared with previous studies. Future research needs to control these variables to determine the reason for these differences in outcome. *See related editorial:* ***Plungers and Polemics: Active Compression-Decompression CPR and Federal Policy****—CM Olson, D Rennie. JAMA 1995;273 (16):1299-1300.*

The Accuracy of Drug Information from Pharmaceutical Sales Representatives—MG Ziegler, P Lew, BC Singer. JAMA 1995;273 (16):1296-1298.

OBJECTIVE: To provide quantitative data about the accuracy of the information about drugs presented to physicians by pharmaceutical sales representatives. DESIGN: One hundred six statements about drugs made during 13 presentations by pharmaceutical representatives were analyzed for accuracy. Statements were rated inaccurate if they contradicted the 1993 *Physician' Desk Reference* or material quoted or handed out by the sales representative. SETTING: University teaching hospital. RESULTS: Twelve (11%) of 106 statements about drugs were inaccurate. All 12 inaccurate statements were favorable toward the promoted drug, whereas 39 (49%) of 79 accurate statements were favorable (P = 0.005). None of 15 statements about competitors drugs were favorable, but all were accurate, significantly ($P < 0.001$) differing from statements about promoted drugs. In a survey of 27 physicians who attended these presentations, seven (26%) recalled any false statement made by a pharmaceutical representative, and 10 (37%) said information from the representatives influenced the way they prescribed drugs. CONCLUSIONS: Eleven percent of the statements made by pharmaceutical representatives about drugs contradicted information readily available to them. Physicians generally failed to recognize the inaccurate statements.

Immunomodulation by Theophylline in Asthma: Demonstration by Withdrawal of Therapy—J Kidney, M Dominguez, PM Taylor, M Rose, KF Chung, PJ Barnes. Am J Respir Crit Care Med 1995;151:1907-1914.

Theophylline is the most widely used anti-asthma drug worldwide and is classified as a bronchodilator, although there is increasing evidence that it may have immunomodulatory effects. We have investigated the effects of theophylline withdrawal under placebo control in 27 asthmatic patients (25 to 70 years) treated with long-term theophylline who were also treated with high dose inhaled corticosteroids. We measured asthma symptoms (diary card), lung function (spirometry and home records of peak expiratory flow), and peripheral leukocyte populations using dual color flow cytometry. In eight of these patients, we examined fiberoptic bronchial biopsies by immunocytochemistry. We also studied peripheral blood lymphocytes in eight asthmatic patients who had never received theophylline. Mean steady state plasma theophylline concentrations during theophylline therapy were 8.6 ± 0.9 mg/L. Theophylline withdrawal was associated with a significant increase in asthma symptoms, particularly at night, and a fall in spirometry and morning peak flow. This was accompanied by a significant fall in peripheral blood monocytes ($CD14^+$, activated $CD4^+$ T-lymphocytes ($CD4^+/CD25^+$) and activated $CD8^+$ T-cells ($CD8^+/HLA\text{-}DR^+$) in patients with a plasma theophylline > 5 mg/L. The lymphocyte populations in theophylline-naïve patients were similar to those found after theophylline withdrawal. Bronchial biopsies showed a mirror image of the peripheral blood with an increase in $CD4^+$ and $CD8^+$ lymphocytes in the airway. Chronic treatment with theophylline, even at low plasma concentrations, controls asthma symptoms and has effects on T-lymphocyte populations in the peripheral blood which are the inverse of those observed in the airways. This indicates that theophylline has immunomodulatory effects and that its place in asthma therapy should be reassessed.

Informed Consent in Emergency Research: Consensus Statement from the Coalition Conference of Acute Resuscitation and Critical Care Researchers—MH Biros, RJ Lewis, CM Olson, JW Runge, RO Cummins, N Fost. JAMA 1995;273(16):1283-1287.

OBJECTIVE: A coalition conference of acute resuscitation researchers was held to discuss the feasibility of applying current federal research regulations regarding informed consent to the emergency setting. This article presents consensus recommendations for regulatory changes for consent in emergency research. PARTICIPANTS: Representatives from the Society for Academic Emergency Medicine and the American Heart Association identified several professional organizations as stakeholders in this issue, including research, clinical, bioethics, legal, and patient advocacy groups. The Office for Protection from Research Risks (OPRR), the Food and Drug Administration (FDA), and staff from specific legislative offices were also invited to observe. Forty-three participants attended, including representatives from 12 professional organizations, 5 medical institutions, and the FDA and OPRR. This was a closed meeting. Participants were self-funded or sponsored by their professional organizations. EVIDENCE: Before the meeting, a draft of a position statement was developed by the conference organizers based on the current literature and discussions with experts in the field. This draft, copies of the current federal research regulations, and supporting articles were distributed before the conference. CONSENSUS PROCESS: Participants rotated through mod-

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erated discussion sessions to comment on subsections of the draft. Following discussion, a working draft was developed and distributed to each participant and represented organizational board for final review. All comments were considered in the final version of the document. CONCLUSIONS: We believe there are circumstances when it is not feasible to obtain prospective or proxy consent for enrollment into an emergency research protocol. In these circumstances, patients are vulnerable, not only to research risks, but also to being denied potentially beneficial therapy when there is no known effective treatment for their life-threatening condition. We offer recommendations that should be met when the critical nature of the illness or injury or the need to apply an investigational therapy rapidly precludes prospective consent for participation in emergency research. *See the related editorial:* ***Research in Emergency Situations: The Role of Deferred Consent****—RJ Levine. JAMA 1995;273(16):1300-1303.*

The Leukotriene D_4-receptor Antagonist, ICI 204,219, Relieves Symptoms of Acute Seasonal Allergic Rhinitis—AL Donnelly, M Glass, MC Minkwitz, TB Casale. Am J Respir Crit Care Med 1995;151:1734-1739.

The efficacy and safety of single oral doses of the leukotriene D_4-receptor antagonist, ICI 204,219, were tested in subjects with acute seasonal allergic rhinitis. Subjects who were enrolled in the double-blind, placebo-controlled trial spent 8 h/day for two consecutive days in a park at the peak of ragweed season (counts > 1,000 grains/m^3). Subjects (n = 164) who had sufficient symptoms during a 3-h baseline period on Day 1 were randomized to treatment with 10 (n = 33), 20 (n = 33), 40 (n = 33), or 100 mg (n = 32) of ICI 204,219 or placebo (n = 33). Rhinitis symptoms (nasal congestion, sneezing, rhinorrhea, itchy nose, throat and palate, and eye symptoms) were recorded hourly in the park and three times each evening at home. Blood samples were collected twice daily to determine plasma levels of ICI 204,219. Nasal congestion improved ($p < 0.01$) most consistently from the evening of Day 1 through Day 2 after treatment with 20- and 40-mg doses of ICI 204,219 versus placebo. Sneezing and rhinorrhea ($p \leq 0.05$) also improved on Day 2 for subjects who received 20- and 40-mg doses of ICI 204,219 compared with placebo. Mean symptoms scores for the entire day showed that 20 mg of ICI 204,219 was the minimally effective dose in this trial. The onset of action for all treatment groups, including placebo, was within the first 2 h of dosing. No serious adverse events were reported during the trial. ICI 204,219 was well tolerated and relieved symptoms of acute seasonal allergic rhinitis.

Association of Young Maternal Age with Adverse Reproductive Outcomes—AM Fraser, JE Brockert, RH Ward. N Engl J Med 1995;332: 1113-1117.

BACKGROUND: Pregnancy in adolescence is associated with an excess risk of poor outcomes, including low birthweight and prematurity. Whether this association simply reflects the deleterious sociodemographic environment of most pregnant teenagers or whether biologic immaturity is also causally implicated is not known. METHODS: To determine whether a young age confers an intrinsic risk of adverse outcomes of pregnancy, we performed stratified analyses of 134,088 white girls and women, 13 to 24 years old, in Utah who delivered singleton, first-born children between 1970 and 1990. Relative risk for subgroups of this study population was examined to eliminate the confounding influence of marital status, educational level, and the adequacy of prenatal care. The adjusted relative risk for the entire study group was calculated as the weighted average of the stratum-specific risks. RESULTS: Among white married mothers with educational levels appropriate for their ages who received adequate prenatal care, younger teenage mothers (13 to 17 years of age) had a significantly higher risk ($P < 0.001$) than mothers who were 20 to 24 years of age of delivering an infant who had low birthweight (relative risk, 1.7; 95 percent confidence interval, 1.5 to 2.0), who was delivered prematurely (relative risk, 1.9; 95 percent confidence interval, 1.7 to 2.1), or who was small for gestational age (relative risk, 1.3; 95 percent confidence interval, 1.2 to 1.4). Older teenage mothers (18 or 19 years of age) also had a significant increase in these risks. Even though sociodemographic variables associated with teenage pregnancy increase the risk of adverse outcomes, the relative risk remained significantly elevated for both younger and older teenage mothers after adjustment for marital status, level of education, and adequacy of prenatal care. CONCLUSIONS: In a study of mothers 13 to 24 years old who had the characteristics of most white, middle-class Americans, a younger age conferred an increased risk of adverse pregnancy outcomes that was independent of important confounding sociodemographic factors.

Growth of Prepubertal Children with Mild Asthma Treated with Inhaled Beclomethasone Dipropionate—IJ Doull, NJ Freezer, ST Holgate. Am J Respir Crit Care Med 1995;151: 1715-1719.

Poorly controlled severe asthma can lead to growth impairment in childhood. In children with mild asthma, it is less clear whether treatment influences growth or adrenal function. We determined in a randomized, double-blind, placebo-controlled, community-based study, the effect of inhaled beclomethasone dipropionate (BDP) 400 μg/day for 7 months on the linear growth and adrenal function of 94 children 7 to 9 years of age. Height was measured at least monthly during treatment, and adrenal function was assessed by overnight urinary cortisol at baseline and after 3 and 6 months of treatment. Mean regressed daily growth was significantly decreased during the treatment period in the BDP-treated group, 0.79 versus 1.14 mm/wk (difference 0.35 mm/wk; 95% CI −0.46 to −0.25; $p < 0.0001$). At the end of the 7 months, the BDP-treated children had grown significantly less than the children on placebo: mean of 2.66 versus 3.66 cm (difference 1.0 cm; 95% CI −1.36 to −0.64 cm; $p < 0.0001$). Growth was significantly decreased in both males and females. During a washout period of 4 months, there was no significant catch-up growth. BDP had no effect on overnight urinary cortisol production. BDP, at a dose taken by many children, significantly decreases statural growth in children with mild asthma, and this effect is unlikely to be mediated through the hypothalamo-pituitary-adrenal axis.

Randomized Prospective Trial of Noninvasive Positive Pressure Ventilation in Acute Respiratory Failure—N Kramer, TJ Meyer, J Meharg, RD Cece, NS Hill. Am J Respir Crit Care Med 1995;151:1799-1806.

Recent studies suggest that noninvasive positive pressure ventilation (NPPV) administered by nasal or oronasal mask avoids the need for endotracheal intubation, rapidly improves vital signs, gas exchange, and sense of dyspnea, and may reduce mortality in selected patients with acute respiratory failure, but few controlled trials have been done. The present study used a randomized prospective design to evaluate the possible benefits of NPPV plus standard therapy versus standard therapy alone in patients with acute respiratory failure. Patients to receive NPPV were comfortably fitted with a standard nasal mask connected to a BiPAP ventilatory assist device (Respironics Inc, Murrysville PA) in the patient flow-triggered/time-triggered (S/T) mode, and standard therapy consisted of all other treatments deemed necessary by the primary physician, including endotracheal intubation. The need for intubation was reduced from 73% in the standard therapy group (11 of 15 patients) to 31% in the NPPV group (5 of 16 patients, $p < 0.05$). Among chronic obstructive pulmonary disease (COPD) patients, the reduction was even more striking, with 8 of 12 (67%) control patients requiring intubation compared with 1 of 11 (9%) NPPV patients ($p < 0.05$). Heart and respiratory rates were significantly lower in the NPPV group than in control patients within 1 h, and P_{aO_2} was significantly improved in the NPPV group for the first 6 h. Dyspnea scores and maximal inspiratory pressures were better in the NPPV than in control patients at 6 h, and nurses and therapists spent similar amounts of time at the bedside for both groups. Duration of ventilator use, hospital stays, mortality rates, and hospital charges were also similar. Nasal NPPV was well tolerated, and complications were few and minor. We conclude that nasal NPPV reduces the need for intubation in patients with acute respiratory insufficiency who are otherwise stable, particularly those with COPD.

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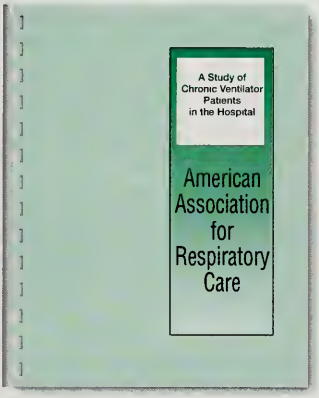

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Trends in Cigarette Smoking among US Adolescents, 1974 through 1991—DE Nelson, GA Giovino, DR Shopland, PD Mowery, SL Mills, MP Eriksen. Am J Public Health 1995; 85(1):34-40.

OBJECTIVES: The purpose of this study was to determine national trends in adolescent cigarette smoking prevalence. METHODS: We conducted trend analyses based on 1974 through 1991 current smoking prevalence data among persons aged 12 through 19 years from the National Household Surveys on Drug Abuse, High School Seniors Surveys, and National Health Interview Surveys. RESULTS: Overall smoking prevalence declined much more rapidly from 1974 through 1980 (1.9 percentage points annually among younger adolescents; the range among surveys of older adolescents was 0.2 to 2.0 percentage points annually) than from 1985 through 1991 (0 to 0.5 percentage points annually among all adolescents). Since 1980, smoking has generally declined at a slightly faster rate among older female adolescents than among male adolescents. Smoking among Black adolescents of all ages declined in nearly every survey population during each study period (range among surveys: 1974-1985 = 1.0 to 2.9 percentage points; 1985-1991 = 0.7 to 1.5 percentage points annually); for White adolescents, only minimal declines in smoking have occurred since 1985. CONCLUSIONS: Since 1974, major changes in adolescent smoking patterns have occurred, especially among Blacks. The overall slowing rate of decline in smoking prevalence since 1985 may indicate success of increased tobacco advertising and promotional activities targeted at adolescents or inadequate antitobacco education efforts.

Facial Wrinkling in Men and Women, by Smoking Status—VL Ernster, D Grady, R Miike, D Black, J Selby, K Kerlikowske. Am J Public Health. 1995;85(1):78-82.

OBJECTIVES: This study examined the association of smoking status and pack-years of smoking with facial wrinkling in men and women. METHODS: We conducted a cross-sectional study of 299 never smokers, 551 former smokers and 286 current smokers, aged 30 through 69 years, drawn from a health maintenance organization. Smoking status, pack-years of smoking, and potential confounding variables were assessed by questionnaire. Facial wrinkle category, a dichotomous variable, and facial wrinkle score, a computed continuous variable, were assessed by blinded standardized visual assessment. Wrinkling was so uncommon among 30- through 39-year-old subjects that analyses were restricted to subjects aged 40 and over (227 never smokers, 456 former smokers, and 228 current smokers). RESULTS: With age, average sun exposure, and body mass index controlled, the estimated relative risk of moderate/severe wrinkling for current smokers compared to never smokers was 2.3 (95% confidence interval [CI] = 1.2, 4.2) among men and 3.1 (95% CI 1.6, 5.9) among women. Pack-years was positively associated with facial wrinkle score in women aged 40 through 69 years and in men aged 40 through 59 years. In both groups, the increased risk of wrinkling was equivalent to about 1.4 years of aging. CONCLUSIONS: Our results support earlier findings that risk of facial wrinkling is greater in cigarette smokers than in never smokers.

Pentamidine Aerosol Versus Trimethoprim-Sulfamethoxazole for *Pneumocystics carinii* in Acquired Immune Deficiency Syndrome—AB Montgomery, DW Feigal Jr, F Sattler, GR Mason, A Catanzaro, R Edison, et al. Am J Respir Crit Care Med 1995;151:1068-1074.

Pneumocystis carinii pneumonia remains one of the most common opportunistic infections in patients with acquired immune deficiency syndrome (AIDS). Treatment with either intravenous pentamidine or trimethoprim-sulfamethoxazole (TMP-SMX) is frequently complicated by serious adverse reactions. This study was a prospective, blinded comparison of 600 mg/d of pentamidine as an aerosol versus 15 mg/kg/d of trimethoprim plus 75 mg/kg/d of sulfamethoxazole for patients with mild or moderately severe *P carinii* pneumonia (alveolar arterial oxygen difference of less than 55 mm Hg). Of 367 participants who were randomized to receive study therapies, 287 had proven and 16 had presumed *Pneumocystis* pneumonia. There were 29 deaths within 35 d of study initiation: 12 in the aerosolized pentamidine group and 17 in the TMP-SMX groups (log rank p = 0.28). The difference in mortality was 3.4% (95% CI = −3.5, 10.8%). Ninety-four patients treated with aerosolized pentamidine had to have their study therapy changed because of lack of efficacy, compared with 22 patients treated with TMP-SMX (p = 0.002). In addition P_{aO_2} improved faster in patients treated with TMP-SMX. However, aerosolized pentamidine was discontinued less often than TMP-SMX because of toxicity (9.4 versus 40%, p < 0.001). Rash (0.6 versus 14.9%), nausea and vomiting (1.7 versus 12.2%), and abnormalities of liver function tests (1.7 versus 12.2%) were the most common adverse effects necessitating treatment discontinuation. During 6-mo follow-up there was no difference in mortality. Recurrences of *P carinii* pneumonia were more frequent in patients treated with aerosolized pentamidine. Although aerosolized pentamidine was better tolerated than TMP-SMX, patients treated with TMP-SMX showed treatment failure, less often showed more rapidly improved oxygenation, and relapsed less often.

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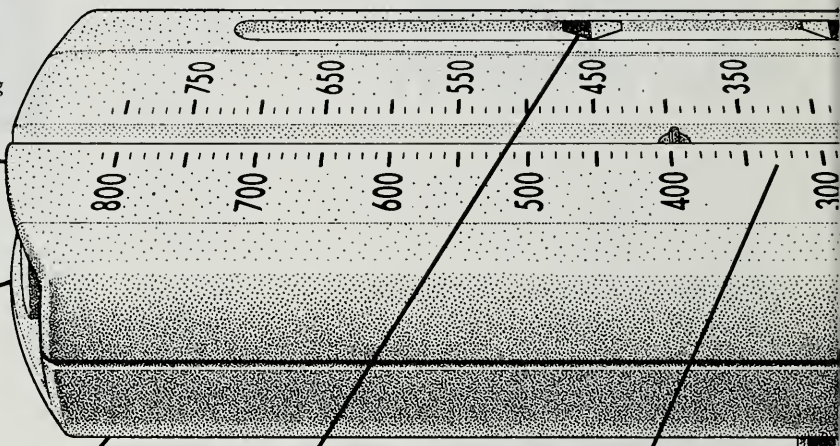

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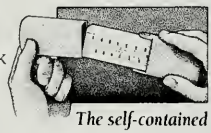

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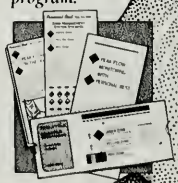

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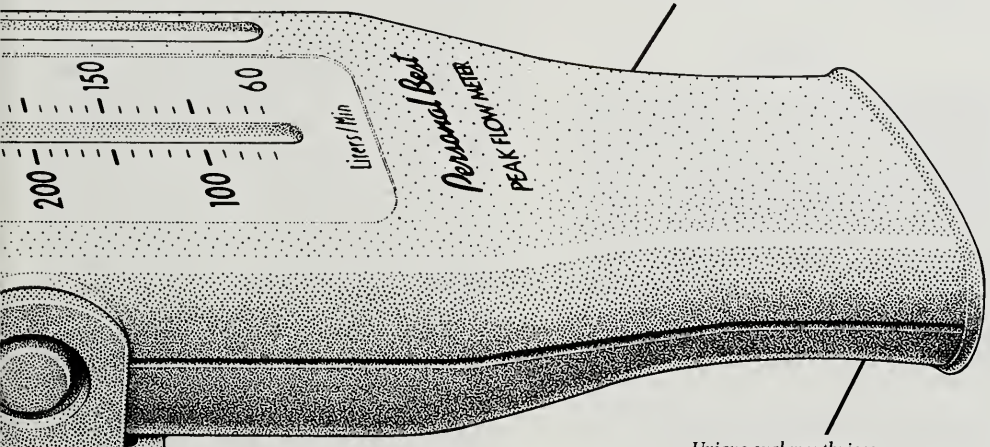

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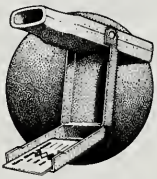

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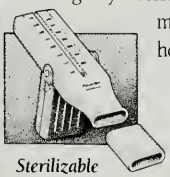

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Respiratory Care in the Era of Work Redesign: Out with the Bath Water (Maybe) but Do Save the Baby!

Introduction

Patient-focused care, one facet of work redesign in the health-care setting, has become an enticing strategy in the current cost-attentive health-care environment.[1] Specifically, by bringing caregivers and services to the patient, patient-focused care attempts to streamline health-care delivery in a way that conserves resources, maximizes coordination of services, and assures maximal patient satisfaction. Like other aspects of health-care reform, patient-focused care has laudable goals that have drawn much attention and have caused a growing number of institutions to launch pilot studies investigating its effectiveness. However, whether these laudable goals have been met awaits results of much needed studies. In the meantime, patient-focused care may have important implications for the roles of respiratory care practitioners (RCPs) in inpatient care. For example, designers of patient-focused care models must address several questions about respiratory care practitioners:

- Will RCPs be a component of the patient-care team in the patient-focused-care model?
- If so, what will be the actual role of RCPs? Specifically, will RCPs be cross-trained and interchange roles with all other health-care providers, or will therapists focus exclusively on the patient's respiratory care needs?
- Can other members of the patient-focused care team be cross-trained to provide the less-complex respiratory care treatments, allowing therapists to focus on aspects of patient-care requiring respiratory expertise?

Because little attention has been given to these issues, my purpose in writing this editorial is to consider the role of respiratory therapists in patient-focused care models.

A main goal of patient-focused care is to coordinate caregivers' activities while capitalizing on the respective expertise of each component member of the patient care team. To the extent that respiratory therapists have unique expertise in assessing and treating respiratory conditions, successful patient-focused care models will preserve this role for respiratory therapists and avoid the risks of diluting this expertise by cross-training less expert caregivers for specialized respiratory tasks. Several lines of evidence argue for preserving this specialized role for therapists in patient-focused care: (1) Respiratory therapists can be better allocators of diagnostic and therapeutic respiratory services than other health-care providers.[2,3] (2) Misallocation of respiratory care activities is common currently.[4] (3) A Respiratory Therapy Consult Service using therapist-driven protocols (now more appropriately called patient-driven protocols) can lessen misallocation of respiratory care while reducing costs.[5] Let us consider each of these points.

First, studies have shown that respiratory therapists can be better allocators of diagnostic and therapeutic respiratory services than are other health-care professionals.[2,3] For example, in several studies comparing therapists to both nurses and physicians, RCPs have demonstrated a better fund of knowledge and better demonstration skills using a variety of inhaler devices.[6-8] In a study examining arterial-blood-gas ordering practices, Browning et al[3] showed that therapists were less likely to order arterial blood gases inappropriately than were other health-care providers in a surgical ICU. In another study in which therapists made decisions in weaning cardiothoracic surgical patients from mechanical ventilation, RCPs weaned patients with success equal to that of managing physicians and in a shorter time.[9] Regarding use of aerosolized medications and bronchial hygiene techniques, Hart et al[2] showed that therapists' interaction with physicians lessened the frequency of inappropriate orders for these respiratory services. Overall, as briefly summarized here (but reviewed elsewhere),[10] current evidence strongly endorses the unique role of respiratory therapists in determining and administering respiratory therapy. In this regard, optimal work redesign models will take maximal advantage of current excellence by preserving therapists' roles as assessors and implementers of specialized respiratory care services.

A second line of evidence that work redesign should preserve a specialized role for therapists is that therapist-driven protocols improve respiratory care. As recently reviewed in this journal,[10] reports of successful implementation of therapist-driven protocols have come from many institutions, including Lutheran Medical Center (Wheat Ridge, Colorado), the University of Michigan (Ann Arbor, Michigan), the University of California at San Diego, Fox Chase Cancer Center (Philadelphia, Pennsylvania), Memorial Mission Hospital (Asheville, North Carolina), and the Cleveland Clinic Foundation (Cleveland, Ohio). The effectiveness of these protocols provides evidence for an expanded role for RCPs.

Encouraged by others' experience with therapist-driven protocols, we have compared Respiratory Therapy Con-

sult Service-directed respiratory care to physician-directed care at the Cleveland Clinic Foundation and found that despite managing patients with more severe respiratory illness, the Respiratory Therapy Consult Service prescribes more appropriate respiratory care at similar treatment costs and without adverse sequelae.[5] Experience with therapist-driven protocols also suggests that successful implementation requires skilled therapists, a receptive hospital, a supportive medical director, and active quality management to assure standardized training and implementation of protocols. In this regard, it is clear that successful use of therapist-driven protocols requires excellent respiratory therapists with strong assessment and communication skills, high motivation, and a thirst for innovation. Indeed, these characteristics are hard enough to find, and easy substitution by 'cross-trained' alternate caregivers seems very unlikely. Thus, although work redesign may allocate some simpler respiratory care tasks (eg, administering incentive spirometry, measuring and recording pulse oximetry saturations) to nontherapists—if evidence of equivalent clinical and cost-effectiveness become clear—the advantages of preserving the therapist's key role as assessor of respiratory care needs seems clear.

In summary, although work redesign is an exciting new strategy for delivering health care, 'hard-nosed' examination of the work-redesign model is needed before conclusions about its advisability are possible. We should ask: Should we proverbially "throw out the bath water" and change the current time-honored structure of delivering care for widespread work redesign? Given the infancy of the idea and available studies, the answer is "maybe," or better yet "let's wait and see." However, while we are building and studying work redesign models, it makes sense to preserve the benefits of existing models of respiratory care. Lest the baby be jettisoned with the bath water, patient-focused care models should preserve the role of respiratory therapists as assessors and deliverers of specialized respiratory care services.

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Cleveland, Ohio

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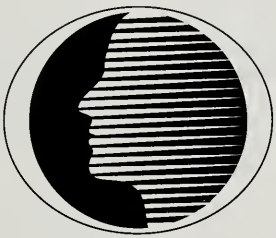

FACE TO FACE WITH CHANGE

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Inhaled Nitric Oxide Delivery Systems: A Role for Respiratory Therapists

Introduction

Inhaled nitric oxide (NO), a selective pulmonary vasodilator, may be useful in the treatment of acute respiratory distress syndrome (ARDS) and other lung diseases characterized by pulmonary hypertension and hypoxemia.[1-3] Although investigational, inhaled NO is currently receiving intense clinical and laboratory study. Multi-institutional trials are underway to evaluate the usefulness of inhaled NO in neonates with persistent pulmonary hypertension and adults with ARDS. At the 1995 meeting of the American Thoracic Society, 264 abstracts related to nitric oxide were presented compared to only 148 abstracts dealing with mechanical ventilation.

Although it is likely to be several years before inhaled NO is approved for general use, respiratory therapists should become familiar with issues related to this therapy. Because it is administered by inhalation, delivery systems for NO legitimately fall within the practice of respiratory therapists. In this issue of RESPIRATORY CARE, Betit et al describe their delivery system for infants.[4] At our institution, we use a neonatal NO delivery system similar to that described by Betit et al.[4] Consistent with their results, we have also found that calculated and measured NO concentrations differ.[5]

The Ideal NO Delivery System

There are no commercially available NO delivery systems, and the devices used vary considerably from institution to institution. Betit et al[4] list the characteristics of an ideal delivery system for inhaled NO during mechanical ventilation. In this editorial, we would like to expand their list based upon our experience with NO therapy in more than 100 neonatal, pediatric, and adult patients during the past 5 years. The duration of treatment for these patients has varied from several hours to more than 2 months.

Be dependable and safe: Inhaled NO is used with the most critically ill patients. Complex systems may be more likely to permit errors that could compromise ventilation, oxygenation, or delivery of the correct NO concentration ([NO]). The function of NO delivery systems must be thoroughly evaluated in the laboratory before patient use. These evaluations should be published to assist others.

Deliver precise and stable NO doses: For adults, inhaled NO is usually administered at concentrations ≤ 20 ppm. Doses as high as 80 ppm may be occasionally used, and doses > 80 ppm are not indicated. Concentrations that are too high can cause complications such as methemoglobinemia and production of unacceptable levels of nitrogen dioxide (NO_2). Our unpublished observations suggest that an inadvertent low dose can cause rebound hypoxemia and pulmonary hypertension. Thus it is important to deliver a precise and stable dose.

Limit NO_2 production: Although the Occupational Safety and Health Administration (OSHA) has set safety limits for NO_2 at 5 ppm,[6] airway reactivity and parenchymal lung injury have been reported with NO_2 concentrations ([NO_2]) as low as 2 ppm,[7,8] Our clinical goal is to maintain inhaled [NO_2] as low as possible (always < 2 ppm). The extent of conversion of NO to NO_2 is determined by the residence time of NO with O_2 and is accelerated by increased [NO] and high fractional concentration of delivered oxygen (F_{DO_2}). In adult mechanical ventilators, the residence time is determined by the volume of the system and by the minute ventilation.[9] At NO doses ≤ 20 ppm, the risk of significant NO_2 production in adult ventilator systems is low unless the F_{DO_2} is very high (≥ 0.9) and the minute ventilation is very low (≤ 5 L/min). In continuous flow systems such as those used in pediatrics, [NO_2] remains low because of the short residence time. Although the use of soda lime has been recommended to limit NO_2 delivery,[10,11] NO_2 generation can be minimized by proper system design.[9] Further, placement of a soda lime canister into the circuit increases resistance, increases the likelihood of leaks, increases compressible volume, modifies inspiratory flow waveforms, and may affect triggering.

Monitor NO and NO_2: Many clinical centers that are administering inhaled NO have developed nomograms to assist with proper dosing. As illustrated in the paper by Betit et al,[4] these calculations may not agree precisely with the measured dose. This is likely due to imprecision of the metering devices used to mix NO, N_2, and O_2—none of which have been calibrated for use with NO. Therefore, it is important to directly monitor inhaled [NO]. It is also necessary to monitor inhaled [NO_2] because of its potential for harm.

Permit scavenging of NO: Concerns regarding contamination of the environment with NO and NO_2 and the potential for adverse effects on health-care providers at the bedside are legitimate. OSHA has set exposure limits for NO at a time-weighted average of 25 ppm.[6] This is higher than the typical NO dose (≤ 20 ppm). In intensive care unit environments that have > 6 air exchanges/h, ambient NO levels should remain low. It has been our experience that ambient NO levels are low during NO administration (< 0.25 ppm) with or without scavenging. Nonetheless, we do scavenge exhaled gases during NO therapy. It is important that the scavenging system be constructed so that it does not affect the function of the ventilator and does not increase expiratory resistance. It should be appreciated that scavenging gases from the exhalation port of the ventilator does not completely eliminate ambient contamination because the ventilator may internally leak gas (containing NO) as part of its normal operation.

Maintain proper ventilator function: Adapting the ventilator to deliver NO should not affect its function. In particular, the alarm systems should not be affected. The addition of NO lowers the F_{DO_2}, and for that reason oxygen concentration should be monitored distal to the site of NO titration into the system. There is concern regarding the effect of NO on the internal components of the ventilator and the external blenders and flowmeters that are exposed to NO; however, we have detected no damage or malfunction of equipment related to NO exposure with our system, after thousands of hours of exposure.

Techniques for NO Delivery during Mechanical Ventilation

NO is typically supplied by a cylinder in a high concentration (800-2,200 ppm). It is then diluted with N_2, air, or oxygen before delivery to the patient. The choice of cylinder concentration is determined by safety and convenience. A higher concentration is convenient (fewer cylinder changes) but increases the risk of exposure to high [NO] for both the patient and bedside care providers. Betit et al[4] describe their method for delivery of NO with a continuous-flow pediatric ventilator. Other systems have been described that are applicable to other ventilators, and each has advantages and disadvantages.

Titration into the Inspiratory Limb

For continuous-flow ventilators, such as those used in pediatrics, NO can be titrated into the inspiratory limb of the ventilator circuit. NO should be titrated into the system near the ventilator outlet for adequate mixing before reaching the patient. This should keep NO_2 generation low because the residence, or dwell, time in the system is short. The expected [NO] can be calculated as:

$$[NO] = \frac{(\text{NO flow})(\text{source ppm})}{\text{NO flow} + \text{ventilator flow}}$$

However, this is an approximation and the actual delivered [NO] should be measured. Once the [NO] has been established, it should remain constant provided that the total flow through the system does not change.

We do not recommend such systems for use with adult ventilators that have phasic flow. With such systems, the inspiratory circuit fills with NO during expiration, and a large bolus of NO is then delivered to the patient with the beginning of each breath.[12] NO delivery is also affected by the inspiratory flow waveform, by changes in minute ventilation, and by the site at which NO is titrated into the circuit. Also, the additional flow augments tidal volume and makes triggering difficult.

Titration at the Ventilator Y-piece

If NO is added at the Y-piece,[13-15] the inspiratory circuit does not fill with NO during exhalation. Instead, the NO bleeds out through the expiratory limb of the ventilator during exhalation. Most important, it is not possible to measure the inspired [NO] with this method, and the dose can only be approximated by mathematical calculation. This system suffers many of the same limitations of NO titration into the inspiratory circuit (as described above).

Injection into the Trachea

Another method that has been used is the administration of a constant flow of NO via a catheter passed into the trachea. Problems associated with this method are similar to titration at the Y-piece. During apnea, O_2-free NO gas continues to flow into the trachea. This can quickly produce hypoxia, and we believe the method is dangerous.

Injection into the Circuit during Inspiration

NO may be injected into the ventilator circuit during the inspiratory phase,[3,16,17] by using a nebulizer drive mechanism that operates during inspiration. The gas supply to the nebulizer contains the [NO] required to achieve the desired patient dose after mixing with gas delivered from the ventilator. Because the gas flow from the nebulizer is constant during inspiration, this method delivers a constant [NO] dose with constant-flow ventilation. However, it does not work well with varying inspiratory flow patterns, such as pressure control. Also, this method does not deliver a stable dose with ventilatory modes such as pressure support in which tidal volume and inspiratory time vary from breath to breath.

The major benefit of this method is that it minimizes NO_2 generation because the residence of NO in an O_2-containing

gas mixture is minimized. However, this method does not allow precise control of the inspired [NO] and is only available with a few ventilator systems. For this method to be acceptable, flow from the ventilator must be continuously and precisely measured, and the injected dose of NO must be precisely titrated so that the delivered [NO] and inspiratory flow waveform are not affected.

Premixing before Introduction

We administer NO to adult, mechanically ventilated patients by premixing the NO with N_2 (or air) and introducing the mixture into the high-pressure air inlet of the ventilator.[6,18,19] An air/oxygen blender is used to add NO (ie, 800 ppm) to the O_2-inlet of the blender while N_2 or air is added to the air inlet of the blender. The choice of air or N_2 as the diluent is determined by the extent of NO_2 generation. NO must be mixed with N_2 with high delivered NO doses (> 20 ppm), high F_{DO_2} (eg, > 0.90 for the Puritan-Bennett 7200 and > 0.75 with Servo 900C), low minute ventilation (eg, < 5 L/min with the Puritan-Bennett 7200 and < 10 L/min with the Servo 900C), or a large internal volume of the ventilator system (eg, Servo 900C).[6] The gas mixture leaving the blender is delivered to the high-pressure air inlet of the ventilator. The final [NO] delivered to the patient is determined by the F_{DO_2} settings on the external blender and the ventilator. Although we have developed nomograms to assist the respiratory therapist at the bedside in determining the correct blender setting to achieve the desired [NO], we always confirm the delivered [NO] by direct measurement.

The advantages of this system are that the NO dose is constant throughout inspiration and is not affected by changes in minute ventilation or flow waveform. However, this system can result in high [NO_2] if appropriate precautions are not observed, and there is a greater NO_2 production with systems that have large internal volumes.[6] Although a potential exists for damage to the blender and ventilator due to the NO with this system, we have detected no damage even after thousands of hours of inhaled NO therapy. One potential disadvantage of this system is that the NO dose changes when the ventilator F_{DO_2} setting is changed. However, it is easy to compensate for this by adjusting the setting on the external blender.

Mixing within the Ventilator

Current-generation ventilators use microprocessor technology to set F_{DO_2}. It seems reasonable to use similar technology to control the delivery of other gases such as NO. Microprocessor technology coupled with precision solenoids could be used to mix air, O_2, and NO to achieve the desired F_{DO_2} and [NO]. However, it is unlikely that industry will release such systems unless inhaled NO is approved by the Food and Drug Administration (FDA) for general use by inhalation.

NO & NO_2 Analysis

Regardless of the method used to deliver NO, it is mandatory to monitor the delivered dose. It is also desirable to monitor NO_2 because of its toxicity. Betit et al[4] show that the calculated dose may differ from the actual delivered dose—an observation that is consistent with our experience and that makes analysis mandatory. The principal methods used to monitor [NO] and [NO_2] are chemiluminescence and electrochemical analysis.

Chemiluminescence vs Electrochemical Analysis

Chemiluminescence is the established method for NO and NO_2 analysis. It has been used for many years in industrial and environmental applications and has been adapted for biomedical uses. Chemiluminescence analyzers are precise; however, they are also expensive and cumbersome to use, and their accuracy may be affected by high concentrations of O_2, N_2, H_2O, and CO_2. Electrochemical cells have recently become available to measure both [NO] and [NO_2]. These are designed specifically for medical applications and are small, portable, rugged, and less expensive than chemiluminescent devices. Evaluations of these devices have found them suitably accurate for clinical use.[20,21] Some electrochemical cells are affected by humidity, and they should, therefore, sample gas before it is humidified. These cells are also pressure sensitive; this effect can be decreased by use of sidestream sampling techniques.

Most chemiluminescent and electrochemical analyzers have slow response times. Thus, the displayed [NO] and [NO_2] are time-weighted averages. This is important to appreciate because real-time fluctuations in NO delivery will not be detected using these methods. This can result in a deceptive impression of the characteristics of the NO delivery pattern and dose.

Inspired-, Tracheal-, & Expired-Gas Analysis

Most commonly [NO] is measured in the inspiratory limb of the ventilator circuit. This location accurately reflects the [NO] delivered to the lungs. [NO] has also been measured in tracheal gas. However, tracheal [NO] is affected by changes in expiratory [NO] and by the ventilatory pattern. Inhaled [NO] should be measured and reported in a reproducible manner. To compare the results of clinical studies, we believe that [NO] should be analyzed from the inspiratory limb of the ventilator circuit.

Conclusion

Respiratory therapists should work with physicians and engineers to develop reliable NO delivery systems for mechanically ventilated patients. As Betit et al[4] have done, it is important that NO delivery systems be systematically evaluated in the laboratory before they are used with patients. The

best method for administering inhaled NO to mechanically ventilated patients is yet to be determined. This should be an important area of research for respiratory therapists. Different methods for delivery and analysis of NO may produce differing results. Thus, it is important that investigators clearly describe their methodology for delivery and analysis of NO.

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Inhaled Nitric Oxide: Evaluation of a Continuous Titration Delivery Technique for Infant Mechanical and Manual Ventilation

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BACKGROUND: The application of inhaled NO for the treatment of pulmonary hypertension and respiratory failure is an important development. We evaluated NO delivery by an infant ventilator (time-cycled, IMV mode) connected to a test lung, with NO titrated into the circuit upstream from the humidifier. METHODS: V_T, airway pressures, and F_{DO_2} were measured and NO and NO_2 analyzed at baseline and at various NO flows, at 3 F_{DO_2} levels and at a ventilator flow of 8 L/min. NO_2 conversion with 80 ppm NO and F_{DO_2} setting 1.0 was assessed at 3 ventilator flows. The effect of a scavenging device, the sensitivity of the low-airway-pressure alarm (LAP), and a manual ventilation system were also evaluated. RESULTS: Measured NO levels (NO_{meas}) were compared to calculated values (NO_{calc}) in 126 samples ranging from 2.5 to 80.9 ppm. Correlations of NO_{meas} and NO_{calc} were strong at F_{DO_2} 0.3, $r = 0.993$, $p < 0.001$; at 0.6, $r = 0.995$, $p < 0.001$; and at 1.0, $r = 0.993$, $p < 0.001$. NO_{calc} underestimated NO_{meas} with a bias of 4.86 ± 3.77 ppm (13.5 %) and precision of 3.91 and 5.81 ppm. NO_2 did not significantly decrease when flow was increased. Airway pressures and V_T remained stable; F_{DO_2} fell 0.01-0.08 over the range of NO flows. Scavenging did not alter baseline pressure. The LAP did not signal when 3-mm ID tubing was disconnected but did with disconnection of 5-mm tubing. Exhaled NO and NO_2 were effectively eliminated with the manual system ($NO = 0.015 \pm 0.05$ ppm; $NO_2 = 0.553 \pm 0.31$ ppm). CONCLUSIONS: Accurate NO doses can be delivered by this method. NO_{calc} does not replace NO analysis. Production of NO_2 is unchanged when ventilator flow is increased from 8 to 12 L/min. NO doses $\leq$ 40 ppm ensure that NO_2 remains < 2.1 ppm. If a dose > 40 ppm is needed and $F_{DO_2} > 0.95$ required, then a higher NO_{source} is required, and toxic levels of NO_2 may result. It is feasible to deliver and scavenge NO and NO_2 during manual ventilation. These systems may permit the precise and safe delivery of inhaled NO. [Respir Care 1995;40(7):706-715]

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Background

The application of inhaled nitric oxide (NO) for the treatment of pulmonary hypertension and respiratory failure is an important development in critical care.[1,2] The respiratory care practitioner with expertise in the management of mechanical ventilation and administration of medical gases can play a major part in the continuing development of inhaled NO therapy.[3] We describe the evaluation of a simplified NO delivery system applicable to infants.

The biology of endogenous NO has been described in detail.[4,5] The salient points include the formation of NO in vas-

cular endothelial cells from L-arginine and molecular oxygen in a reaction catalyzed by NO synthase.[4,5] NO activates soluble guanylate cyclase and raises smooth-muscle cyclic guanosine monophosphate levels, leading to relaxation of vascular smooth muscle.

It is postulated that in pulmonary hypertension the production of NO from endothelial cells is impaired, and the ensuing vasoconstriction may be countered by delivering inhaled NO.[6] NO delivered by inhalation diffuses from the alveolar space into adjacent vascular smooth muscles and causes relaxation.[6,7] The intravascular action of NO is limited by its affinity for hemoglobin.[4,5] When NO is inhaled, the vasoactive influence is confined largely to the pulmonary vasculature and systemic vasodilation is avoided.[6,7]

The selective and potent pulmonary vasodilation of inhaled NO has been used effectively, both diagnostically and therapeutically, for infants with pulmonary hypertension associated with congenital heart disease and following cardiovascular surgery.[8,9] It may acquire an important role in the management of persistent pulmonary hypertension of the newborn, adult respiratory distress syndrome, and lung transplantation.[10-13]

Although the therapeutic use of inhaled NO is of major clinical importance, improper delivery is not without the potential for toxic effects.[14-16] For instance, methemoglobin is formed when NO binds to hemoglobin; nitrogen dioxide (NO_2) forms when NO reacts with oxygen, and both methemoglobin and NO_2 may be harmful.[14-16] An awareness of these potential toxic effects is important in the development of a delivery system.

An ideal delivery system for the administration of inhaled NO should be of simple design, deliver and verify a wide range of precise NO doses, monitor NO_2 levels while minimizing its accumulation, and permit scavenging of exhaled gases.[1,17] The system should be adaptable to different clinical situations, and, when used in conjunction with mechanical ventilation, should not interfere with ventilator functions, in particular the alarm systems. Ensuring these characteristics should be a priority in the development of an inhaled NO delivery system.

Several delivery systems have been reported.[17] Although these systems provide accurate and safe delivery of inhaled NO, they require the use of two blenders and a third gas source (nitrogen) to obtain the desired NO doses. An empirically derived table of blender settings for specific NO doses has been established by analysis of NO levels with a chemiluminescence device.[17] This equipment, when mounted on a portable cart, is readily adaptable to several clinical situations but remains relatively cumbersome.

The purpose of our study was to evaluate a simplified delivery system, employing continuous titration of NO and, thus, eliminating the need for a second blender, while providing precise doses of NO. The delivery system was designed to be used during mechanical and manual ventilation of infants ($\leq$ 12 kg) because it is in this group that inhaled NO is applied most often at our institution.

Description of Delivery Systems

An NO regulator was attached to source tanks of medical-grade NO containing concentrations of 800 ppm and 2,200 ppm, balanced with nitrogen (N_2) and containing < 8 ppm and 20 ppm NO_2, respectively*. Two oxygen flowmeters, one calibrated in 100 mL/min increments (range 100-1,000 mL/min), and one calibrated in 25 mL/min increments (range 25-200 mL/min) were connected to the regulator, which was adjusted to 50 psi. The regulator was purged 6 times by pressurizing the regulator and slowly releasing the gas to a wall vacuum source. This minimizes residual NO_2 accumulation in the body of the regulator.

Supply tubing was connected to the flowmeter and to a delivery port located at the outlet of an infant ventilator. A 12-inch connection hose, with two sampling ports, was connected to the inlet of a humidifier. An oxygen analyzer was placed in the first port and the sample line for sidestream NO analysis was placed in the second port. This was identified as the distal sampling site.

A standard disposable ventilator circuit with a 10-mm internal diameter (ID) was connected to the outlet of the humidifier. A second sampling port for proximal airway NO analysis was placed 12 inches from the patient connection in the inspiratory tubing. The ventilator circuit and proximal airway flow sensor (pressure-differential pneumotachometer) were connected to a test lung with a compliance of 1.0 mL/cm H_2O. A tidal volume (V_T) monitor, with digital and graphic displays, commercially available with this ventilator, received and processed signals from the proximal airway flow sensor.

A customized scavenging device was affixed to the outer aspects of the exhalation valve. The scavenging device consisted of an evacuation line attached to regulated wall vacuum, a one-way valve permitting the continuous draw of ambient air, and a reservoir bag that would inflate in the event of vacuum failure. Figure 1 illustrates this delivery system.

Following our observation that certain patients receiving prolonged NO therapy do not tolerate interruptions in NO delivery (for instance, during transport) we modified an anesthesia-type resuscitation bag (Mapleson-D configuration) and undertook an investigation to ensure its safety. Based on the premise that NO can be titrated into the main ventilator flow, a second gas supply line was connected into the O_2 supply line of the anesthesia-type resuscitation bag. The scavenging of exhaled gases was facilitated by connecting the adjustable pressure-relief valve to a canister of Purafil® air purification medium via adapted exhaust tubing. Purafil is a scavenging material that binds and inactivates NO and NO_2. Additional sampling ports were placed distal to the gas flow at the patient connection and were used to monitor NO, NO_2, and F_{DO_2}.

* Products and suppliers are listed in the Product Sources section at the end of the text.

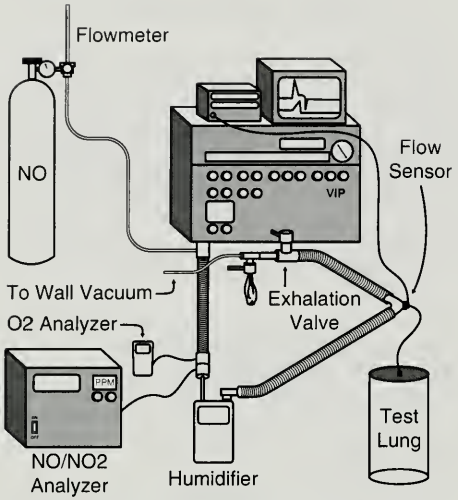

Fig. 1. System for continuous titration of nitric oxide (NO) for infant mechanical ventilation, with sidestream chemiluminescence analyzer.

A sample port was placed between a 12-inch reservoir and the outlet of the canister to monitor NO and NO_2 concentrations at the exhaust port. Figure 2 illustrates this system.

Evaluation Methods

The time-triggered, pressure-limited, time-cycled continuous-flow mode (ie, time-cycled IMV) was selected on the ventilator. Baseline ventilator settings were adjusted to a peak inspiratory pressure (PIP) of 30 cm H_2O, positive end-expiratory pressure (PEEP) 5 cm H_2O, frequency (f) 25 cycles/min, inspiratory time (t_I) 0.6 s, and flow ($\dot{V}$) 8 L/min.

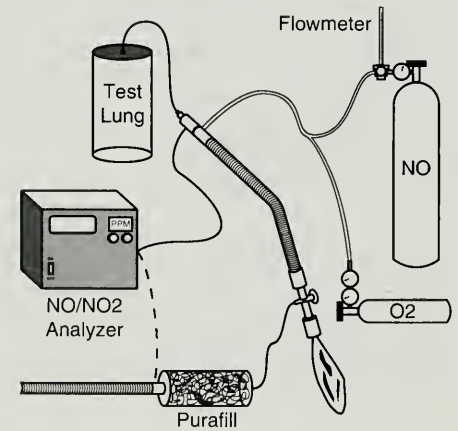

Fig. 2. System for continuous titration of nitric oxide (NO) for infant manual ventilation, with sidestream chemiluminescence analyzer and exhaled gas scavenger.

Digital measurements of ventilator settings and graphic display of $\dot{V}$, proximal airway pressure, and V_T were recorded prior to and following the addition of the scavenging device. PEEP levels were varied from 3-6 cm H_2O to assess the effect of the scavenging device on the ventilator exhalation mechanism.

Various concentrations of NO were introduced into the described delivery systems and measured by a chemiluminescence analyzer equipped with a molybdenum converter.[17-19] Prior to the introduction of NO, a 2-point calibration was completed with the chemiluminescence and oxygen analyzers.

Calculated values for delivered NO were predicted from the equation

$$NO_{calc} = (NO_{source})\left(\frac{\dot{V}_{NO}}{\dot{V}_{total}}\right),$$

where NO_{calc} is the predicted NO concentration delivered in ppm, NO_{source} is the concentration of NO in the source tank in ppm, $\dot{V}_{NO}$ is the set flow from the source in mL/min, and $\dot{V}_{total}$ is the total flow in mL/min—the sum of the $\dot{V}_{NO}$ and the flow from the ventilator, which was set at 8 L/min.

NO_{calc} was established for each flow increment available, using the 800- and 2,200-ppm source tanks (Table 1).

Table 1. Calculated NO Levels (NO_{calc}) at each Flow Increment for Both Source Tanks (800 and 2,200 ppm NO).

800 ppm source		2,200 ppm source	
$\dot{V}_{NO}$ (mL/min)	NO_{calc} (ppm)	$\dot{V}_{NO}$ (mL/min)	NO_{calc} (ppm)
25	2.5	25	6.9
50	5.0	50	13.7
75	7.4	75	20.4
100	9.9	100	27.2
200	19.5	125	33.8
300	28.9	150	40.5
400	38.1	175	47.1
500	47.1	200	53.7
600	55.8	300	79.5
700	64.4		
800	72.7		
900	80.9		

NO and NO_2 concentrations were analyzed at each flow increment and at F_{DO_2} settings of 0.30, 0.60, and 1.0. To determine whether additional mixing of gases occurred in the ventilator circuit, NO and NO_2 were analyzed at both the proximal airway and distal sampling sites. Following 5 minutes of stabilization, analyzed NO and NO_2 levels and F_{DO_2} were recorded. Measured PIP, PEEP, mean airway pressure ($\bar{P}_{aw}$), and V_T were recorded also.

To determine the effect of the ventilator flow on the residence, or dwell, time of NO and O_2 and the resultant accumulation of NO_2, 12 additional NO and NO_2 measurements were obtained. This was accomplished by delivering 80 ppm of NO at a set F_{DO_2} of 1.0, with ventilator flows of 8, 10, and 12 L/min and with both 800 and 2,200 ppm sources. Six measurements were made from each sampling site. The NO flow required to achieve 80 ppm at ventilator flows of 10 and 12 L/min was determined by rearranging the above equation to yield:

$$\dot{V}_{NO} = NO_{calc}\left(\frac{\dot{V}_{total}}{NO_{source}}\right)$$

The estimated $\dot{V}_{NO}$ was then entered into the original equation to reflect the true $\dot{V}_{total}$ and obtain a precise NO_{calc}. This was evaluated using the 800- and 2,200- ppm sources (Table 2). NO at 80 ppm and F_{DO_2} of 1.0 were deemed the most extreme concentrations that would be delivered under institutional protocols and that would result in the highest NO_2 concentration Proximal and distal airway measurements of NO, NO_2, and F_{DO_2} were recorded following 5 minutes of stabilization.

Table 2. Calculated NO Levels (NO_{calc}) with Increasing Total Flow ($\dot{V}_{total}$) for Both Source Tanks.

800 ppm source			2,200 ppm source		
$\dot{V}_{NO}$ (mL/min)	$\dot{V}_{total}$ (mL/min)	NO_{calc} (ppm)	$\dot{V}_{NO}$ (mL/min)	$\dot{V}_{total}$ (mL/min)	NO_{calc} (ppm)
900	8900	80.8	300	8300	79.5
1100	11100	79.3	375	10375	79.5
1300	13300	78.2	450	12450	79.5

We evaluated 3- and 5-mm-ID NO supply tubing to assess the sensitivity of the ventilator low-airway-pressure alarm. Tubing disconnection was simulated and pressure waveforms were observed.

NO was then added to the anesthesia-type manual ventilation system. NO at 1 L/min and O_2 at 9 L/min were added from a portable cylinder (F_{DO_2} 1.0). Thus, the $\dot{V}_{total}$ was 10 L/min, which yields a NO_{calc} of 80 ppm. The resistance to flow across the Purafil canister was 0.2 cm $H_2O \cdot min \cdot L^{-1}$.

With the manual ventilation system connected to the test lung, NO and NO_2 concentrations were analyzed at the supply inlet and then at the outlet of the canister. Levels were recorded every 30 minutes during a 6-hour period. Manual ventilation was intermittently simulated at an approximate frequency of 30 to 40 cycles/min with inflation pressures of 25-30 cm H_2O and baseline pressures of 5-8 cm H_2O, while analyzed NO levels were observed.

The comparison of calculated and measured NO levels was evaluated by determining the correlation coefficient and by analysis of linear regression. Additionally, bias and precision between calculated and measured NO values were obtained to determine their degree of agreement. The differences between proximal and distal NO and NO_2 levels were also evaluated by obtaining estimated mean differences and 95% confidence intervals (CI). Measurements corresponding to increases in $\dot{V}_{total}$ were evaluated by regression analysis. The means and standard deviations for monitored ventilator variables were established, and the mean values for exhaled NO and NO_2 levels were estimated and 95% CI established. Statistical analysis was accomplished with commercially available software.

Evaluation Results

Digital measurements and graphic display of ventilator settings demonstrated no change with the addition of the customized scavenging device; no change in baseline waveforms occurred when PEEP levels were increased from 3 to 6 cm H_2O.

NO and NO_2 levels were obtained from 126 samples, 63 at the proximal airway site and 63 at the distal site. Of these 63 pairs of samples, 36 were obtained with the 800 and 27 with the 2,200 ppm NO. Measured NO concentrations ranged 2.8-90.6 ppm, and NO_2 levels ranged from 0.24 to 8.0 ppm. NO_2 progressively increased with both concentrations supplied but always remained < 2.1 ppm when NO concentrations were < 40 ppm, at all F_{DO_2}s and with both NO concentrations (Figs. 3A and 3B).

NO measurements obtained from the distal site were slightly higher than from the proximal airway site, with an estimated mean difference of 1.59 ± 1.61 ppm [95% CI 1.18 ppm, 1.99 ppm]. Distal NO_2 measurements were also slightly higher than proximal with an estimated mean difference of 0.297 ± 0.498 ppm [95% CI 0.172 ppm, 0.423 ppm]. A two-way analysis of variance was used to determine whether these estimated mean values differed across varying levels of F_{DO_2} and NO. This analysis determined that the estimated mean values were not affected by these factors or their interaction ($p = 0.51$).

Analyzed distal NO levels ($n = 63$) were compared to calculated values at: F_{DO_2} 0.3, $r = 0.993$, $p < 0.001$; F_{DO_2} 0.6, $r = 0.995$, $p < 0.001$; and F_{DO_2} 1.0, $r = 0.993$, $p < 0.001$ (Fig. 4). Measured NO was consistently higher than NO_{calc}, with a mean difference or bias of 4.86 ± 3.77 ppm (13.5 %) and precision (± 0.95) from 3.91 to 5.81 ppm (Fig. 5). This was consistent at each F_{DO_2}.

As F_{DO_2} was increased for each NO flow, there was a slight decrease in analyzed NO. This was most evident at NO flow > 400 mL/min (ie, NO levels > 40 ppm) with NO levels analyzed at 1.2% to 8% lower as F_{DO_2} was increased.

Monitored ventilator variables remained stable throughout the study, PIP 31.5 ± 0.4 cm H_2O, PEEP 5 ± 0 cm H_2O, $\bar{P}_{aw}$ 11.5 ± 0.2 cm H_2O, V_T 25.6 ± 0.4 mL.

As the NO flow was increased, the analyzed F_{DO_2} became progressively less than the set F_{DO_2}, as expected. For example, with a $\dot{V}_{NO}$ set at 25 mL/min the analyzed F_{DO_2} was 0.01 lower,

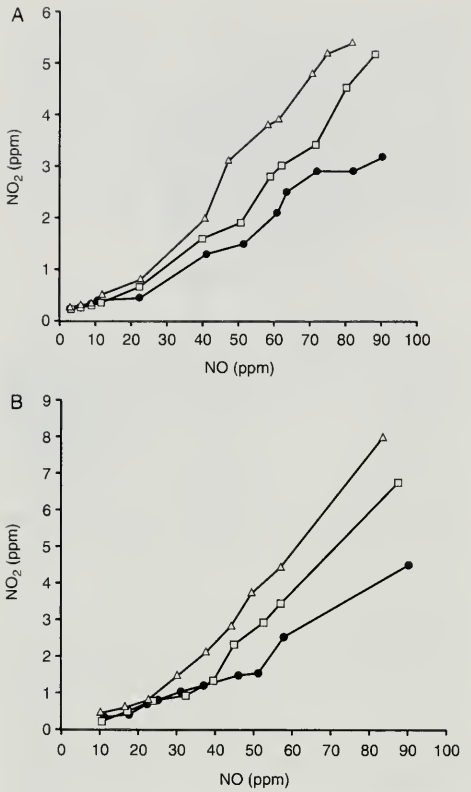

Fig. 3. A. Nitrogen dioxide (NO_2) accumulation with increasing levels of nitric oxide (NO) with an 800 ppm source tank. B. Nitrogen dioxide (NO_2) accumulation with increasing levels of nitric oxide (NO) with a 2,200 ppm source tank.

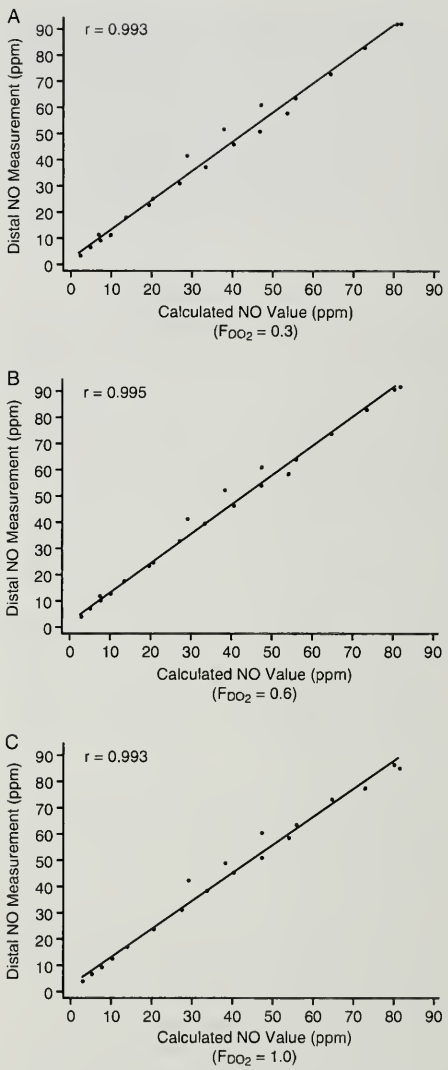

Fig. 4. Correlation between calculated nitric oxide (NO) values and distal NO measurements at F_{DO_2} settings of (A) 0.3, (B) 0.6, and (C) 1.0.

in contrast to an NO flow set at 900 mL/min at which the analyzed F_{DO_2} was 0.06-0.08 lower.

In the 12 additional NO and NO_2 measurements obtained to assess the dwell (ie, residence) time of NO and O_2, regression analysis demonstrated that increasing total flow does not appear to decrease NO_2 levels significantly with either source concentration of NO, (800 ppm source $p = 0.234$, 2,200 ppm source $p = 0.073$).

Disconnection of the 5.0-mm-ID tubing resulted in a loss of PEEP and successful activation of the low-airway-pressure alarm. This alarm failed when the supply tubing with a 3.0-mm ID was disconnected. However, disconnection of either size tubing from the adapter resulted in successful activation of the low-airway-pressure alarm.

The NO levels at the supply inlet of the transport system were 92.1 ± 0.05 ppm. The estimated mean of the NO levels that were measured at the outlet of the canister were 0.015 ± 0.047 ppm [95% CI –0.015 ppm, 0.045 ppm]. Analyzed NO_2 levels at the supply inlet were 9.6 ± 1.2 ppm, with an estimated mean level at the canister outlet of 0.553 ± 0.311 ppm [95%

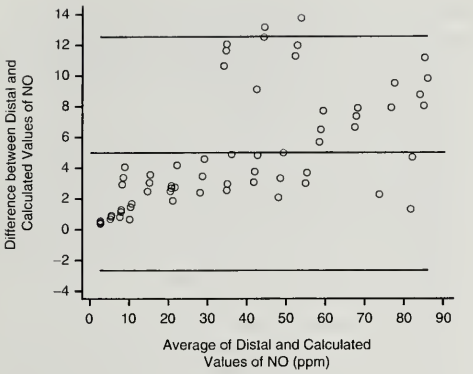

Fig. 5. The limits of agreement between NO levels measured distally and calculated NO levels (–2.70 ppm and 12.42 ppm).

CI 0.356 ppm, 0.750 ppm]. NO and NO_2 levels did not change when manual ventilation was simulated, and F_{DO_2} remained stable at 0.92.

Discussion

Inhaled NO has been delivered in a number of clinical trials and in conjunction with several devices, but primarily with mechanical ventilators.[17-22] NO and NO_2 levels can be measured by electrochemical and chemiluminescence analyzers.[17-25] During the development of delivery systems, problems inherent to both types of analyzers were noted, which raised concerns regarding the accuracy of delivered NO doses and NO_2 levels.[23-25,30]

We have demonstrated that NO can be titrated in conjunction with an infant ventilator supplied with continuous flow. Because there is a strong correlation between calculated and measured NO levels, the equation,

$$NO_{calc} = NO_{source}\left(\frac{\dot{V}_{NO}}{\dot{V}_{total}}\right),$$

can be used to estimate NO doses and determine initial flow requirements.

The underestimation of NO_{meas} by NO_{calc} may be due to imprecise adjustments of NO flow. We did not verify any of the flowrates because this is not typically performed in the clinical setting. Adjustments were made by visualizing the markings of the flowmeter. Because oxygen flowmeters were used and their calibration does not account for the density of an NO/N_2 mixture, flowmeter inaccuracy may also have contributed to this underestimation.

If large discrepancies exist between analyzed and calculated levels, the accuracy of the analyzer may be questioned, which may prompt the user to recalibrate it or inspect the delivery system for other problems such as leaks. One problem that may occur is the introduction of the demand flow that is available with the ventilator used in this study. If the baseline pressure falls by 1 cm H_2O the demand valve opens and introduces additional flow into the circuit. If the demand flow is activated intermittently and for short periods, the response of the NO analyzer may not be rapid enough to detect a subtle change in the NO level. If the demand flow display is observed frequently and for prolonged periods, the clinician should assess the adequacy of the ventilator flow and determine whether the dilution of the NO dose has an adverse physiologic effect.

We have incorporated into our protocols the practice of comparing calculated with analyzed NO levels. This provides quality control to aid practitioners in identifying and troubleshooting technical and user-associated problems. However, the NO_{calc} does underestimate the measured level and should not be used exclusively to determine NO doses. NO doses and NO_2 levels should be analyzed.

Additional mixing of gases in the ventilator circuit appears to occur as evidenced by the slight difference between the distal and proximal airway sites (1.59 ± 1.6 ppm). This slight difference may not be clinically important unless low doses (ie, < 3 ppm) are required. Because the difference between proximal and distal NO_2 levels is small (0.29 ± 0.49), distal measurements provide an accurate assessment of NO_2 production. The ability to obtain accurate NO and NO_2 measurements from the distal site (ie, upstream from the humidifier) is valuable because both chemiluminescence and electrochemical analyzers may be susceptible to inaccuracy in the presence of humidity.[16,20,29]

Chemiluminescence is currently (1995) considered the gold standard and is the standard by which most medical-grade NO sources are verified. However, at present, this form of analysis is cumbersome and expensive and designed primarily for industrial use. These drawbacks are being addressed, and the next-generation chemiluminescence analyzers, modified for clinical use, will be provided with alarms and be more portable.

Another limitation of chemiluminescence analysis is the quenching phenomenon. Quenching, is the underestimation of NO due to the presence of other gases and water vapor.[17,23,29,30] O_2 in particular influences this phenomenon, which is more pronounced as higher concentrations of O_2 are introduced.[17,29] Quenching has been reported to result in a maximum underestimation of NO by 10%.[29] Recently, chemiluminescence analyzers have been equipped with molybdenum converters, which minimize the quenching phenomenon and promote stable NO_2 readings.[15,20,26,30]

The quenching effect was demonstrated by a slight decrease in NO readings as F_{DO_2} was increased. This slight decrease ranged from 1.2%, when NO was approximately 40 ppm, to as high as 8.0%, with 80 ppm of NO. If exact measurements

are critical, a correction factor can be used.[29] Because NO doses used clinically have been generally < 40 ppm, the need to correct for quenching does not seem crucial.

The accumulation of NO_2 is dependent on the concentrations of NO and O_2 and the amount of time that these gases are in contact with the circuit.[25-27] This is referred to as the residence, or dwell, time.[25-27] It has been demonstrated that NO_2 accumulation can occur in 12-36 seconds at 80 ppm of NO and at an F_{DO_2} of 0.95.[25,26] Our study demonstrates that no significant change in the production of NO_2 occurs as the ventilator flow is increased from 8 L/min to 12 L/min. Therefore, a flow of 8 L/min is sufficient to minimize NO_2 production. Flows > 12 L/min may further reduce the dwell time but may result in turbulent expiratory flow, leading to increased work of breathing, particularly with small-bore ventilator circuits. A ventilator circuit with a larger ID would accommodate a higher flow, but the increased volume of the circuit might further contribute to the dwell time of NO and O_2.

Our understanding of the effect that NO_2 levels, even below 5 ppm, have on the lungs is limited.[15] The toxicology of NO_2 is complex and appears to vary with the conditions of the local airway environment.[15] Inhalation of NO_2 at doses as low as 2 ppm has been associated with terminal bronchiole epithelial hypertrophy and alveolar cell hyperplasia in animals.[15] The local airway environment (eg, temperature and pH) may alter the effects of the nitrogen oxides.[15] NO_2, a product of grain fermentation, has been implicated in the human occupational disease known as silo filler's disease, which is associated with pulmonary edema, hemorrhage, and bronchiolitis obliterans.[15]

We initially observed a maximum NO_2 of 8.0 ppm with the mechanical ventilator delivery system and a mean ± SD of 9.6 ± 1.2 ppm with the manual ventilation technique. We were concerned about these high NO_2 levels and were prompted to repeat these measurements. Subsequent measurements yielded a maximum NO_2 of 5.7 ppm and a mean ± SD of 6.5 ± 1.1 ppm with the mechanical and manual ventilation systems respectively. These NO_2 levels occurred only with NO doses > 60 ppm and at a F_{DO_2} of > 0.91.

The variability in NO_2 levels that we observed, further supports the need to closely monitor NO_2 levels during inhaled NO delivery. We have not typically observed this variability during clinical use because lower NO doses are used, and the production of NO_2 is small. The high NO_2 levels and the variability that we observed may be influenced by several factors.

The chemical reaction between NO and O_2 may not be uniform throughout the ventilator circuit. The reaction may occur much more rapidly at the point at which the NO mixture from the cylinder enters the ventilator circuit and mixes with the ventilator flow. The 2,200 ppm mixture may produce a much more rapid reaction than the 800 ppm mixture. It is also possible that the turbulent flow characteristics may cause the kinetics of the molecules to be highly variable. As a result, the NO_2 may not be fully purged near the delivery site and may even accumulate in the corrugations of the tube that connects the ventilator with the humidifier. This connecting tube has a volume of 85 mL, and the distance between the delivery site and the sample site is 12 inches.

Additional NO_2 production may occur in the sample line of the chemiluminescence analyzer. The length of the sample line and the sample flow influence the dwell time of NO and O_2. If additional NO_2 were produced in the sample line, the reading on the analyzer would not reflect the NO_2 of the ventilator circuit alone. The sample line length was 4 feet, the ID was 1/8 in., and a low sample flow of 100 mL/min was used.

Another source of NO_2 may be the NO tanks. The source tanks used in this study contained approximately 1% NO_2. This NO_2 may actually be contained in the walls of the cylinders and may be inconsistently released during delivery. There may also be a layering effect with NO_2 concentrations being higher as the tank approaches empty.

Inhaled NO is titrated to the lowest possible NO dose that achieves the desired physiologic response. In general, doses < 40 ppm and most often even < 20 ppm have been clinically effective. Our delivery system ensures that NO_2 levels are < 2.1 ppm at NO dose ≤ 40 ppm and for any F_{DO_2}.

There may be circumstances in which it is critical to use higher NO and O_2 concentrations, and under which the favorable physiologic responses of these doses outweigh their potential toxic effects. We have had to employ this strategy in 5 infants receiving NO via the mechanical ventilation system. In these infants a maximum NO dose of 80 ppm and F_{DO_2} of 0.96 were used. Under these conditions, we observed a mean ± SD NO_2 level of 5.7 ± 2.1 ppm.

The manual ventilation system may have higher NO_2 levels because of the presence of a partially distended reservoir. If it is critical to transport a patient with NO, the risks of delivering high NO_2 concentrations with this system must be carefully considered.

Placing soda lime in the ventilator circuit has been suggested as a means of minimizing or even eliminating NO_2 accumulation.[18,27] The amount of soda lime required has not been fully investigated, and a large container of soda lime may affect circuit resistance and compressible volume, which are of particular concern with pediatric patients. We are evaluating the use of soda lime in these delivery systems so that safe administration may be ensured when high NO and O_2 concentrations are required.

The industrial standard for environmental exposure to NO (as established by the American Conference of Governmental Industrial Hygienists, or ACGIH and the Occupational Safety and Health Association, or OSHA) is a time-weighted average (TWA) of 25 ppm.[28] The ACGIH has established a TWA and short-term exposure limits (STEL) of 3 and 5 ppm respectively for NO_2.[28] OSHA has set a STEL of 1 ppm and a ceiling of 5 ppm for NO_2.[28]

Because NO_2 levels were < 1.0 ppm at NO levels ≤ 25 ppm at all F_{DO_2}, and below the ACGIH and OSHA limits, scavenging may not be necessary. However, when doses of NO > 25 ppm are used, NO_2 levels can be > 1 ppm and scavenging should be established in order to avoid the STEL and ceiling exposure limits for both NO and NO_2. Because NO is generally delivered for prolonged periods and scavenging of exhaled gases can easily be accomplished, we have adopted the practice of scavenging all exhaled gases during NO delivery, regardless of the dose being used. NO and NO_2 levels should also be periodically assessed in the immediate patient care environment particularly if leaks around the patient's endotracheal tube exist.

This study was performed with a test lung, and it is not known whether the NO_2 levels obtained are similar to those measured clinically. Because analysis occurs in the inspiratory limb of the circuit, it is unlikely that the patient's pulmonary mechanics significantly influence NO_2 measurements with this system.

The analyzed F_{DO_2} was noted to be less than the set F_{DO_2} as the NO flow was increased. This accounts for the dilutional effect that the N_2 balance of the source tanks has on the F_{DO_2}. During clinical application, this could be remedied by increasing the set F_{DO_2} and continuously analyzing it.

The dilution of F_{DO_2} would only be a problem if an F_{DO_2} of 1.0 were critical. If it is critical to deliver NO doses at > 40 ppm and at $F_{DO_2} > 0.95$, then a higher NO_{source} will be needed.

Based on the equation

$$\left(\frac{\dot{V}_{NO}}{\dot{V}_{total}}\right) 100 = \%\ N_2,$$

the maximum F_{DO_2} with a set 'F_{IO_2}' of 1.0 can be determined. For example, the maximum F_{DO_2} that can be achieved with a NO_{calc} of 80.9 ppm, drawn from an 800 ppm NO source, is 0.90 but the maximum F_{DO_2} for a dose of 79.5 ppm dose using a 2,200 ppm NO source is 0.96. This is due to the difference in flowrates required for each. The use of the 800 ppm NO concentration would require a flowrate of 900 mL/min whereas a NO concentration of 2,200 ppm requires only 300 mL/min.

Based on the calculations described, a table of predicted NO doses delivered at various ventilator flowrates and from sources of various NO concentrations can be established. The maximum F_{DO_2} attainable at each dose may also be included (Table 3). Such information provides a reference point for flow adjustments and reduces the chance of miscalculations.

Table 3. Predicted NO Doses at Various NO Flows, Ventilator Flows, and NO Concentrations, and Maximum Attainable O_2 Derived from the Equations $NO_{calc} = \dot{V}_{NO} / \dot{V}_{total} \cdot NO_{source}$ and $\%N_2 = \dot{V}_{NO} / \dot{V}_{total} \cdot 100$. Part A: NO_{source} = 800 ppm. Part B: NO_{source} = 2,200 ppm.

		Ventilator Flow = 8 L/min		Ventilator Flow = 10 L/min		Ventilator Flow = 12 L/min	
	NO_{source} (mL/min)	NO_{calc} (ppm)	Max O_2 (%)	NO_{calc} (ppm)	Max O_2 (%)	NO_{calc} (ppm)	Max O_2 (%)
A	25	2.5	99.7	2.0	99.8	1.7	99.8
	50	5.0	99.4	4.0	99.5	3.3	99.6
	75	7.4	99.1	6.0	99.3	5.0	99.4
	100	9.9	98.8	7.9	99.0	6.6	99.2
	150	14.7	98.2	11.8	98.6	9.9	98.8
	200	19.5	97.6	15.7	98.1	13.1	98.4
	300	28.9	96.4	23.3	97.1	19.5	97.6
	400	38.1	95.3	30.8	96.2	25.8	96.8
	500	47.1	94.2	38.1	95.3	32.0	96.0
	600	55.8	93.1	45.3	94.4	38.1	95.3
	700	64.4	92.0	52.3	93.5	44.1	94.5
	800	72.7	91.0	59.3	92.6	50.0	93.8
	900	80.9	90.0	66.1	91.8	55.8	93.1
	1000	89.0	89.0	72.7	91.0	61.5	92.4
B	25	6.9	99.7	5.5	99.8	4.6	99.8
	50	13.7	99.4	10.9	99.5	9.1	99.6
	75	20.4	99.1	16.4	99.3	13.7	99.4
	100	27.2	98.8	21.8	99.0	18.2	99.2
	125	33.8	98.5	27.0	98.8	22.7	99.0
	150	40.5	98.2	32.5	98.6	27.2	98.8
	175	47.1	97.9	37.8	98.3	31.6	98.6
	200	53.7	97.6	43.1	98.1	36.1	98.4
	300	79.5	96.4	64.0	97.1	53.7	97.6
	400	104.7	95.3	84.6	96.2	71.0	96.8
	500	129.4	94.2	104.8	95.3	88.0	96.0

The addition of the various adapters and flows does not interfere with the normal functions of this ventilator. Airway pressures and tidal volume delivery were consistent and remained unchanged over a wide range of NO flows. The customized scavenging device, when attached to the outer aspects of the exhalation valve, does not affect this mechanism and maintains baseline pressures. These modifications ensure additional safety to both patient and caregiver.

Because chemiluminescence analyzers are not currently equipped with alarms, high and low oxygen alarms should be used to identify any inadvertent changes in the NO flow. A low-oxygen alert indicates that the NO flow has been increased (ie, additional N_2 and NO are being drawn from the source tank). Conversely, a high alarm may indicate that the NO flow has been reduced or discontinued.

We found that it is important to use supply tubing with an ID $\geq$ 5.0 mm in order to ensure that the disconnect alarm remains sensitive to decreases in airway pressure. We use standard suction tubing for this purpose and have found standard oxygen tubing to be inadequate. The supply tubing should also provide a snug fit when connected to the NO flowmeter. It is also important to utilize adapters with delivery ports that have a minimum internal diameter of 5.0 mm in the event that the tubing becomes disconnected from these sites. The effects that NO and NO_2 have on the components used with this ventilator are unknown; however, with the exception of the exhalation valve, all components are disposable.

Delivery of NO through an anesthesia-type resuscitation bag is feasible and scavenging of exhaled gases can be effectively accomplished. Because the chemiluminescence analyzer as now constituted is not suited for the transport of patients, especially in a small space, calculated NO levels could be verified prior to departure. Electrochemical analyzers may be more suitable during transport.

Scavenging low doses of NO (eg, < 25 ppm) during brief transports may not be necessary. Because most transports utilize portable E-cylinders containing an F_{DO_2} of 1.0, the production of NO_2 at this F_{DO_2} may be greater, especially during prolonged transports. Scavenging, therefore, seems prudent and is easily accomplished.

This manual ventilation system may be modified for use during endotracheal suctioning when interruptions in NO delivery may not be tolerated.[30] Exhaled gases could be diverted to a regulated wall-vacuum source instead of the Purafil canister. Because pressurization of the anesthesia-type resuscitation bag depends on flowrate and careful regulation of the pressure-relief valve, training with this device prior to use on a patient is critical.

The use of inhaled NO has been investigated at our institution for more than 3 years, and in this time more than 275 patients have been treated under both diagnostic and therapeutic protocols. We have successfully employed the simplified continuous titration technique in 41 patients and have experienced results consistent with this bench evaluation.

Conclusions

Our findings suggest that accurate NO doses can be continuously titrated into the inspiratory limb of an infant ventilator circuit and via an anesthesia-type resuscitation bag. The comparison of calculated and analyzed doses may be useful to determine analyzer or system problems. The systems described can provide safe and accurate delivery during mechanical and manual ventilation and are being utilized in protocols for infants with pulmonary hypertension in our institution. Future developments in analyzers and NO delivery, such as mass flow controllers incorporated into commercially available ventilators, will provide even more precise and compact delivery of NO. The chemistry and characteristics of NO_2 in these delivery systems and methods to minimize it require further investigation.

PRODUCT SOURCES

NO Sources:
800 and 2,200 ppm Nitric Oxide balance Nitrogen Tanks, Scott Specialty Gases, Plumstead PA

Flowmeters:
Classic 200 and Classic 1000 Oxygen Flowmeters, Timeter, St Louis MO

Supply Tubing:
3.0-mm ID: 1115 Oxygen Supply Tubing, Hudson RCI, Temecula CA
5.0-mm ID: Argyle Universal Bubble Tubing, Sherwood Medical, St Louis MO

Ventilator:
VIP Bird, Bird Products Corp, Palm Springs CA

Humidifier:
Conchatherm III Plus, Hudson RCI, Temecula CA

Ventilator Circuit:
Isothermal Infant Circuit, Baxter Healthcare Corp, Deerfield IL

Oxygen Analyzer:
5590 Oxygen Monitor, Hudson RCI, Temecula CA

Test Lung:
Neonatal/Pediatric Test Lung, Infrasonics Inc, San Diego CA

Resuscitation Bag Pressure-Relief Valve:
6025 Safety Relief Valve, Vital Signs, Totowa NJ

Resuscitation Bag:
Customized Anesthesia-type, Technimed, Everett MA

Air Purification Medium:
Purafil,® Purafil Inc, Chamblee GA

NO/NO_2 Analyzer:
42M Chemiluminescence NO-NO_2-NO_x Analyzer, Thermoenvironmental Instruments, Franklin MA

Statistical Software:
Stata, Stata Corp, College Station TX

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Patient-Determined Inspiratory Flow during Assisted Mechanical Ventilation

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BACKGROUND: We wished to determine whether providing patients the capability to exceed the set inspiratory flow during mechanical ventilation is associated with a decrease in work-of-breathing indices. METHODS: A Bird 8400-ST ventilator, modified to assure a minimum but variable tidal volume (V_T) and to allow patient-determined inspiratory flow ($\dot{V}_{pd}$), was used to compare assisted mechanical ventilation (AMV) with a preset inspiratory flow ($\dot{V}_{set}$) to AMV with $\dot{V}_{pd}$. Patients (n = 18) were studied during 3 periods of ventilation—$\dot{V}_{set\text{-}1}$, $\dot{V}_{pd}$, and $\dot{V}_{set\text{-}2}$. The set respiratory rate (f) equaled half the actual assist f. F_{DO_2}, minimum V_T, and set f were held constant. Minute ventilation ($\dot{V}_E$), f, arterial blood-gas (ABG) evaluations, end-tidal CO_2 ($P_{et}CO_2$), O_2 consumption, CO_2 production, cardiac output, heart rate, systemic and pulmonary arterial pressure, pressure-time product (PTP), intrinsic positive end-expiratory pressure ($PEEP_i$), esophageal pressure (P_{es}), peak and mean airway pressure, peak inspiratory flow ($\dot{V}_{max}$), respiratory drive ($P_{0.1}$), patient (WOB_{pt}) and ventilator (WOB_{vent}) work of breathing, and duty cycle (t_I/t_{tot}) were measured. RESULTS: $\dot{V}_{max}$ and $P_{et}CO_2$ were significantly higher with $\dot{V}_{pd}$ than with either $\dot{V}_{set\text{-}1}$ or $\dot{V}_{set\text{-}2}$, and $\dot{V}_E$, f, P_{es} and PTP were lower with $\dot{V}_{pd}$ than with $\dot{V}_{set\text{-}2}$. There were no significant differences in the hemodynamic, ABG, or metabolic data. Although not statistically significant, tendencies were noted toward a reduction in $PEEP_i$, $P_{0.1}$, and WOB_{pt}, and toward an increase in WOB_{vent} with $\dot{V}_{pd}$. CONCLUSION: If allowed, patients exceed the $\dot{V}_{set}$ during AMV, reducing indices of patient work [Respir Care 1995;40(7):716-721]

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This study was performed at the University of Michigan Medical Center and the University of Cincinnati Medical Center. It was presented by Mr Haas at the American College of Chest Physicians Annual Meeting held in Chicago, Illinois, October 1992.

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Introduction

Ideally, patient contribution to the total work of breathing (WOB) during assisted mechanical ventilation (AMV) should be minimal. However, during AMV, the inspiratory muscles remain active throughout the inspiratory cycle in healthy volunteers and in critically ill patients.[1-5] Thus, there are two components of inspiratory work—*ventilator work* (WOB_{vent}) occurring as the ventilator forces gas into the lungs and *patient work* (WOB_{pt}) as the inspiratory muscles draw gas into the lungs. The magnitude of WOB_{pt} depends upon patient variables, (eg, compliance [C], resistance [R], and ventilatory drive) and on ventilator variables, (eg, sensitivity, peak-inspiratory flow [$\dot{V}_{max}$], and, to a lesser, extent tidal volume [V_T]).

Recent work by Sassoon et al[3] and Ward et al[4] shows that the major determinant of patient work during AMV is the $\dot{V}_{max}$. Normally the maximum inspiratory flow available to the patient is preset during AMV. Although the flow pattern may be modified—to a square, decelerating, accelerating, or sinusoidal wave—the patient usually is not allowed to exceed

the set flow ($\dot{V}_{set}$). The pressure-support-ventilation (PSV) mode allows the patient to control or determine the inspiratory flow, within the boundaries of a preset pressure limit, and has been shown to lower inspiratory muscle workload compared to AMV[6-7] or intermittent mandatory ventilation (IMV).[8] Disadvantages of PSV are that V_T varies as the patient's pulmonary compliance and airway resistance(R_{aw}) change and, thus, PSV may not be able to provide total ventilatory support.[9,10] MacIntyre and colleagues[10] recently reported a bench study and Amato et al[11] a clinical study using modes of ventilation in which $\dot{V}_{set}$ matched patient demand (ie, even above $\dot{V}_{set}$) and delivered a guaranteed minimum V_T. Matching or exceeding the patient's demand for flow on a breath-to-breath basis should minimize WOB_{pt}.

Our study examined a mode of ventilation in which a minimum $\dot{V}_{set}$ and minimum V_T were set, but the patient could exceed either or both. Specifically, the purpose of this study was to compare the effects of $\dot{V}_{set}$ with patient determined inspiratory flow ($\dot{V}_{pd}$) during AMV on patient work of breathing, ventilatory mechanics, gas exchange, hemodynamics, and metabolic variables. We hypothesized that allowing patients to exert more control over inspiratory flow during AMV would reduce the work of breathing.

Materials & Methods

The protocol was approved by the Institutional Review Boards of the 2 medical centers. Entrance criteria included (1) ≥ 18 years of age; (2) a ratio of arterial oxygen partial pressure to the fraction of oxygen delivered (P_{aO_2}/F_{DO_2}) between 75 and 325; (3) mechanical ventilation with a set respiratory frequency (f_{set}) > 8 breath/min; (4) spontaneous respiratory effort; (5) an existing indwelling arterial catheter; and (6) signed informed consent. Patients were excluded from study if they (1) required positive end-expiratory pressure (PEEP) > 15 cm H_2O; (2) had diseases that might alter respiratory drive, such as closed head injury or hepatorenal syndrome; (3) had conditions capable of causing a leak in the ventilator system, such as a bronchopleural fistula or a leak around the artificial airway; (4) had conditions that might complicate placement of an esophageal catheter, such as tracheoesophageal fistula, esophageal varices, or esophageal bleeding; and (5) required vasopressors to maintain blood pressure.

All patients were ventilated with a ventilator* that, in addition to the conventional AMV and intermittent mandatory ventilation (IMV) modes, had a modification allowing the patient to exceed $\dot{V}_{set}$. This modification assures a minimum V_T and also allows a larger V_T on patient demand. The inspiratory flow modification can be applied in either the AMV or IMV mode and is referred to as volume-assured pressure support (VAPS). In the VAPS AMV mode, the inspiratory flow begins in a manner similar to a typical pressure support breath, in that it increases to achieve the set inspiratory pressure and to meet patient demand. The main difference in flow delivery between these two modes is that as flow decreases in VAPS, to a predetermined threshold equal to $\dot{V}_{set}$, the machine continues to deliver flow at the preset rate until the target V_T is delivered. If the target V_T is delivered during the initial phase of inspiration, before flow decreases to $\dot{V}_{set}$, the ventilator terminates inspiration. Should the patient desire a flow higher than the $\dot{V}_{set}$, that flow is achievable during any portion of the inspiratory cycle. In this paper, $\dot{V}_{pd}$ means simply that the patient has the ability to exceed the $\dot{V}_{set}$. However, the term $\dot{V}_{pd}$ is inexact because the inspiratory flow of each breath is not totally determined by the patient. The ventilator determines flow if the patient stops inspiring or if the V_T target has not been delivered when flow decreases to the preset flow level.

We compared the conventional AMV mode with the VAPS AMV mode to ensure that the primary difference between methods of ventilation was the flow capability, not differences between AMV and IMV.

Patients were initially observed in the conventional AMV mode. Inspiratory flow was set to maintain adequate time for exhalation. Determination of this flow setting was assisted by observing the expiratory flow display and monitoring for the presence of intrinsic PEEP ($PEEP_i$). The patient's respiratory rate (f) and V_T were observed. Patients were then studied under 3 conditions: (1) the conventional AMV mode — $\dot{V}_{set\text{-}1}$, (2) the modified AMV mode permiting $\dot{V}_{pd}$, and (3) back to the conventional AMV mode—$\dot{V}_{set\text{-}2}$. $\dot{V}_{set}$, V_T, F_{DO_2}, sensitivity, and PEEP were held constant during all 3 phases. To ensure patient triggering, f was set to one half that of the initial AMV observation period and was not changed throughout all 3 study phases. The ventilator was equipped with a nondisposable breathing circuit. Inspired gas was heated to 34°-37° C at the airway.

An esophageal balloon catheter was placed, and proper positioning was verified by esophageal pressure tracings.[12] A flow transducer was placed at the patient's airway. Both sensors were connected to a calibrated respiratory mechanics monitor, which has been previously validated.[13,14]

After a 20-minute stabilization period, measurements were taken every 10 minutes over a 40-minute study period. Five to 10 breaths containing a complete data set from the numeric data printout of the respiratory-mechanics monitor were averaged to provide V_T, f, minute ventilation ($\dot{V}_E$), peak inspiratory pressure (PIP), $PEEP_t$, $\dot{V}_{max}$, dynamic compliance (C_{dyn}), R_{aw}, WOB_{pt}, WOB_{vent}, esophageal pressure (P_{es}), fraction of inspiratory time or duty cycle (t_i/t_{tot}), transpulmonary pressure generated during the first 100 milliseconds of inspiration ($P_{0.1}$), and pressure-time-product (PTP). Mean airway pressure ($\bar{P}_{aw}$) was measured with a free-standing pressure monitor. Oxyhemoglobin saturation by pulse oximetry

* Manufacturers and suppliers are identified in the Product Sources section at the end of the text.

(S_{pO_2}), end-tidal carbon dioxide (P_{etCO_2}), heart rate, and blood pressure were continuously monitored and recorded at 10-minute intervals. Arterial blood gas analysis was performed at the end of each 40-minute study period. If a pulmonary artery catheter was in place, cardiac output and hemodynamic pressures were also obtained. Exhaled gas was collected and the concentrations of O_2 and CO_2 were analyzed and averaged over 5 minutes, yielding measures of oxygen consumption ($\dot{V}_{O_2}$) and carbon dioxide production ($\dot{V}_{CO_2}$).

After the first 40-minute AMV period ($\dot{V}_{set\text{-}1}$), the ventilator was changed to the VAPS mode. The pressure support control was adjusted so that V_T with VAPS would not exceed V_T with conventional AMV. Following a 20-minute stabilization period, the data were gathered as above. After the 40-minute study period, the conventional AMV mode was reinstituted for the 20-minute-stabilization and the 40-minute-study period ($\dot{V}_{set\text{-}2}$). This last period was to validate the baseline measurements and to identify changes in the values of study variables that may have been due to a change in patient condition. The study was terminated for $SpO_2 < 85\%$, a change in heart rate ± 20%, or a change in blood pressure ± 20%.

Prior to study, we recorded age, gender, diagnosis, P_{aO_2}/F_{DO_2}, endotracheal tube (ETT) size, $\dot{V}_{set}$, $\dot{V}_E$, and duration of ventilatory support.

WOB_{vent} was measured by integrating the airway pressure signal and using the following formula:

$$WOB_{vent} = \int (P_{aw} - P_{atm})\, \dot{V},$$

where P_{aw} = airway pressure; P_{atm} = atmospheric pressure; and $\dot{V}$ = flow.

WOB_{pt} was measured by integrating the negative P_{es} signal during inspiration and calculated using the formula:

$$WOB_{pt} = \int [(P_{es[end]} - P_{es\,[begin]})\, \dot{V} + V_p \left(\frac{V_p}{2}\right) C_{cw}],$$

where $P_{es[end]}$ = end-expiratory esophageal pressure; $P_{es[begin]}$ = esophageal pressure at the beginning of the breath; V_p = patient portion of V_T; and C_{cw} = chest wall compliance (assumed to be 200 mL/cm H_2O).

All work calculations were normalized to volume by the respiratory-mechanics monitor in units of J/L of ventilation. The pressure time product (PTP) is an estimate of metabolic work of the respiratory muscles. It reflects not only the mechanical work of breathing, but also evaluates the isometric forces of respiration. Isometric work does not involve moving a volume of gas and can occur under such conditions as auto-PEEP or with a poorly responsive demand valve. The respiratory-mechanics monitor calculates PTP using a method described by Sassoon et al[15] as the integral of the negative changes in P_{es} during inspiration, taking into account the C_{cw} and t_I.

Statistical analyses were performed using commercially available software. Statistical significance ($p < 0.05$) was determined using repeated-measures analysis of variance (ANOVA). Because of the preliminary nature of our study, we chose a conservative post-hoc analysis (Bonferroni), which was applied to each paired comparison shown to be statistically significant by the ANOVA.[16] Significance for the post-hoc analysis was set at $p < 0.05/3$ or 0.0167.

Results

Seventeen patients were entered into the study, and one patient was studied twice (n = 18). Their mean (± SD) age was 55.8 (21.6) years, C_{dyn} 48.8 (17.9) mL/cm H_2O, R_{aw} 11.1 (6.2) cm $H_2O \cdot s \cdot L^{-1}$, PaO_2/F_{DO_2} 198 (59), F_{DO_2} 0.43 (0.09), and PEEP 5.7 (3.5) cm H_2O. Patients were on the ventilator 8.61(10.9) days (range 0.5-44 days) prior to study. See Table 1 for demographics and baseline data.

Tables 2-5 review the ventilatory parameters and respiratory-mechanics, gas-exchange, and hemodynamic measurements. Statistical significance ($p < 0.05$, ANOVA), was demonstrated for $\dot{V}_{max}$, $\dot{V}_E$, V_T, f, P_{es}, PTP, t_I/t_{tot}, $P_{0.1}$, P_{etCO_2}, pH and S_{aO_2}. Post-hoc analysis revealed that $\dot{V}_{max}$ and P_{etCO_2} were significantly higher with $\dot{V}_{pd}$ than either $\dot{V}_{set\text{-}1}$ or $\dot{V}_{set\text{-}2}$ and that $\dot{V}_E$, f, P_{es} and PTP were significantly lower with $\dot{V}_{pd}$ than $\dot{V}_{set\text{-}2}$. It also demonstrated that pH and S_{aO_2} were higher with $\dot{V}_{pd}$ than with $\dot{V}_{set\text{-}2}$. There were no significant differences in the hemodynamic, or metabolic data. Although not statistically significant, a tendency was noted toward a reduction in $PEEP_i$, $P_{0.1}$, and WOB_{pt}, and an increase in WOB_{vent} with $\dot{V}_{pd}$. No studies were terminated early.

Figure 1 shows the flow and pressure waveforms for a patient during the study. Note that inspiratory flow during the initial portion of $\dot{V}_{pd}$ is higher, compared to $\dot{V}_{set}$.

Discussion

Mechanical ventilation has been thought to unload the respiratory muscles and diminish work of breathing (WOB). Yet, Marini and colleagues[17,18] pointed out that despite high levels of mechanical ventilatory assistance, considerable patient work may still be present. Strategies for reducing patient WOB and alleviating patient-ventilator dyssynchrony include sedation, paralysis, manipulation of ventilator rate, inspiratory flow and I:E, as well as changing the mode of ventilator operation.[17] Identifying the reason for dyssynchrony helps dictate the strategy to be employed.

Using normal volunteers and critically ill patients, Marini et al[1,2,18] found that inspiratory effort continued throughout the delivery of an assisted breath regardless of the sensitivity, $\dot{V}_E$, or $\dot{V}_{set}$. These authors encouraged attempts to match $\dot{V}_{set}$ to the patient's demand for flow, match t_I/t_{tot} with the patient's natural breathing rhythm, and set sensitivity appropriately. Their work demonstrated that WOB_{pt} during assisted ventilation can, in fact, lead to or worsen respiratory muscle fatigue if the patient's effort is poorly matched to the ventilator settings.

Table 1. Demographic Characteristics of Patients Admitted to the Study.

Patient Number/ Hospital	Age (y)/ Gender	P_{aO_2}/F_{IO_2}	$\dot{V}_E$ (L/min)	$\dot{V}_{set}$ (L/min)	ETT (mm ID)	Diagnosis
1/B	52/M	227	11.7	60	8.0	COPD, pneumonia
2/B	55/M	124	12.7	70	8.0	COPD, pneumonia
3/B	74/M	220	14.1	60	8.0	COPD, pneumonia, lung cancer
4/A	63/M	138	10.2	70	8.0	COPD, sepsis, perforated cecum
5/A	70/M	183	12.3	70	7.0	COPD, perforated duodenal ulcer
6/A	62/M	233	14.5	55	8.5	COPD, MVA, pulmonary contusion
7/A	29/M	322	11.0	45	8.0	ARDS, GSW to abdomen
8a/A	20/M	182	16.9	60	8.0	ARDS, sepsis, GSW to abdomen
8b/A		225	11.6	60	8.0	same patient as #8a
9/A	68/M	268	10.8	50	7.5	ARDS, sepsis, aorto-enteric fistula
10/A	38/M	254	14.3	70	8.0	ARDS, pulmonary contusion
11/B	81/M	77	13.6	60	8.0	ARDS, coronary artery bypass
12/A	58/F	180	8.7	60	7.0	sepsis, pneumonia, bowel obstruction
13/B	65/F	180	6.4	40	7.0	sepsis, aorto-femoral bypass
14/B	82/M	156	7.3	60	8.0	sepsis, cellulitis
15/B	76/M	176	7.5	60	8.0	carotid endarterectomy
16/B	74/M	160	13.1	60	8.5	abdominal aortic aneurysm
17/B	18/M	260	11.9	60	7.5	MVA

$\dot{V}_E$, minute ventilation; $\dot{V}_{set}$, inspiratory flowrate set on ventilator; COPD, chronic obstructive pulmonary disease; MVA, motor vehicle accident; ARDS, adult respiratory distress syndrome; GSW, gun-shot wound.

Table 2. Mean (SD) Values of Ventilatory Variables

Variable	$\dot{V}_{set-1}$*	$\dot{V}_{pd}$	$\dot{V}_{set-2}$	p† (ANOVA)
$\dot{V}_{max}$ (L/min)	54.2 (9.6)	62.9 (10.1)	54.9 (10.2)	a, b, c
$\dot{V}_E$ (L/min)	11.8 (3.1)	11.5 (3.2)	12.7 (3.6)	a, c
V_T (mL)	816.0 (22.0)	857.0 (23.0)	812.0 (22.0)	a
f (breath/min)	15.2 (5.6)	14.5 (6.7)	16.5 (6.4)	a, c
$PEEP_i$ (cm H_2O)	2.9 (1.8)	2.5 (2.2)	3.3 (2.8)	0.104
PIP (cm H_2O)	40.2 (10.5)	40.6 (11.9)	40.2 (11.9)	0.884
$\bar{P}_{aw}$ (cm H_2O)	14.9 (5.5)	15.6 (6.0)	15.2 (6.0)	0.256

* $\dot{V}_{set-1}$, ventilator set flow in baseline 1; $\dot{V}_{pd}$, patient-determined flow; $\dot{V}_{set-2}$, ventilator set flow in baseline 2; $\dot{V}_{max}$, peak inspiratory flow; $\dot{V}_E$, minute ventilator; V_T, tidal volume; f, breathing frequency; $PEEP_i$, positive expiratory pressure-intrinsic; PIP, peak inspiratory pressure; $\bar{P}_{aw}$, mean airway pressure.
† a = $p < 0.05$ using repeated-measures analysis of variance (ANOVA); b, c = $p < 0.0167$ for post-hoc analysis, b comparing $\dot{V}_{pd}$ to $\dot{V}_{set-1}$, and c comparing $\dot{V}_{pd}$ to $\dot{V}_{set-2}$.

Table 3. Mean (SD) Values for Respiratory Mechanics Measurements

Variable	$\dot{V}_{set-1}$*	$\dot{V}_{pd}$	$\dot{V}_{set-2}$	p† (ANOVA)
P_{es} (cm H_2O)	12.2 (3.6)	10.7 (4.1)	13.9 (5.4)	a, c
PTP ([cm H_2O s]/min)	73.0 (59.0)	47.0 (49.0)	92.0 (78.0)	a, c
C_{dyn} (mL/cm H_2O)	64.7 (54.8)	68.4 (65.5)	68.4 (63.4)	0.168
R_{aw} (cm H_2O s L^{-1})	11.0 (6.5)	12.3 (9.1)	11.7 (8.0)	0.175
WOB_{pt} (J/L)	0.52 (0.34)	0.43 (0.70)	0.58 (0.60)	0.079
WOB_{vent} (J/L)	2.01 (0.81)	2.16 (0.80)	2.10 (0.81)	2.288
t_I/t_{tot}	0.30 (0.07)	0.28 (0.07)	0.30 (0.07)	a
$P_{0.1}$ (cm H_2O)	2.27 (1.02)	1.94 (1.31)	2.65 (1.36)	a

* $\dot{V}_{set-1}$, ventilator set flow in baseline 1; $\dot{V}_{pd}$, patient-determined flow; $\dot{V}_{set-2}$, ventilator set flow in baseline 2; P_{es}, esophageal pressure; PTP, pressure time product; C_{dyn}, dynamic compliance; R_{aw}, airways' resistance; WOB_{pt}, patient work of breathing; WOB_{vent}, ventilator work of breathing; t_I/t_{tot}, duty cycle; and $P_{0.1}$, inspiratory pressure at 100 ms into inspiration.
† a = $p < 0.05$ using repeated-measures analysis of variance (ANOVA); c = $p < 0.0167$ for post-hoc analysis, comparing $\dot{V}_{pd}$ to $\dot{V}_{set-2}$.

Ward et al[4] measured WOB in 7 patients who were being ventilated in the AMV mode.They manipulated $\dot{V}_{set}$ between 25 and 65 L/min while maintaining a constant set V_T. In this study, the set f was zero, requiring subjects to trigger each breath. They demonstrated that, at low flows, patient WOB approached 65% of the total inspiratory work. They also found that reducing sensitivity from –2 cm H_2O to –5 cm H_2O resulted in a 50% increase in WOB. These authors concluded that inspiratory flow and sensitivity settings were the major determinants of patient WOB during assisted ventilation.

In a similar study, Sassoon and colleagues[3] elucidated the effects of manipulating $\dot{V}$ and V_T during assist-control ventilation in normal subjects. They set $\dot{V}$ at 40, 60, and 80 L/min and V_T at 100%, 125%, and 150% of spontaneous V_T. Results of this study demonstrated that for a given V_T, WOB decreased as ventilator flow increased. They also found no change in $P_{0.1}$ at different flow settings, suggesting that sensitivity settings and patient respiratory drive play an important role at the initiation of inspiration.

Recent work by Manning and colleagues[19] indicates that

Table 4. Mean (SD) Values for Gas-Exchange Measurements

Variable	$\dot{V}_{set\text{-}1}$*	$\dot{V}_{pd}$	$\dot{V}_{set\text{-}2}$	p† (ANOVA)
P_{etCO_2} (torr)	27.1 (6.1)	28.5 (5.8)	27.2 (6.2)	a, b, c
P_{aCO_2} (torr)	42.1 (5.9)	40.8 (5.7)	41.1 (6.2)	0.221
pH	7.42 (0.05)	7.43 (0.05)	7.42 (0.05)	a, b
S_{pO_2} (%)	95.0 (3.2)	95.7 (2.9)	95.5 (2.7)	0.267
S_{aO_2} (%)	94.3 (3.1)	95.3 (3.1)	95.0 (2.8)	a, b
P_{aO_2} (torr)	80.9 (19.2)	88.9 (22.8)	86.8 (23.7)	0.066
$\dot{V}_{O_2}$ (mL/min)	279 (54)	280 (57)	289 (79)	0.549
$\dot{V}_{CO_2}$ (mL/min)	236 (54)	247 (49)	253 (59)	a, d

* $\dot{V}_{set\text{-}1}$, ventilator set flow in baseline 1; $\dot{V}_{pd}$, patient-determined flow; $\dot{V}_{set\text{-}2}$, ventilator set flow in baseline 2; P_{etCO_2}, end-tidal CO_2; S_{pO_2}, oxyhemoglobin saturation measured by pulse oximetry; SaO_2, oxyhemoglobin saturation measured by CO-oximetry; $\dot{V}_{O_2}$, oxygen consumption; and $\dot{V}_{CO_2}$, CO_2 production.

† a = $p < 0.05$ using repeated-measures analysis of variance (ANOVA); b, c, d = $p < 0.0167$ for post-hoc analysis, b comparing $\dot{V}_{pd}$ to $\dot{V}_{set\text{-}1}$, and c comparing $\dot{V}_{pd}$ to $\dot{V}_{set\text{-}2}$, d comparing Vset-1 to Vset-2.

Table 5. Mean (SD) Values for Hemodynamic Measurements

Variable	$\dot{V}_{set\text{-}1}$*	$\dot{V}_{pd}$	$\dot{V}_{set\text{-}2}$	p† (ANOVA)
HR (beats/min)	89 (15.3)	88 (15.4)	88 (17.6)	0.902
$BP_{systolic}$ (mm Hg)	136 (26)	138 (30)	141 (31)	0.102
$BP_{diastolic}$ (mm Hg)	68 (12)	68 (12)	70 (13)	0.216
$PA_{systolic}$ (mm Hg)	34.6 (9.4)	33.3 (8.7)	34.8 (8.7)	0.298
$PA_{diastolic}$ (mm Hg)	13.0 (4.2)	13.1 (4.6)	13.7 (4.2)	0.414
PCWP (mm Hg)	14.0 (2.2)	14.3 (2.1)	14.7 (1.7)	0.253
C.O. (L/min)	7.0 (2.2)	7.0 (2.2)	7.1 (2.3)	0.675

* $\dot{V}_{set\text{-}1}$, ventilator set flow in baseline 1; $\dot{V}_{pd}$, patient-determined flow; $\dot{V}_{set\text{-}2}$, ventilator set flow in baseline 2; HR, heart rate; BP, blood pressure; PA, pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; C.O., cardiac output.

the sensation of 'air hunger' during mechanical ventilation can occur in spite of a normal chemical drive to breathe and that the mismatch between the desired and delivered inspiratory flowrate can cause breathlessness.

It has been suggested that pressure support ventilation or pressure control ventilation may help reduce WOB, owing to the rapid initial flow.[6,20] Tokioka et al[6] found that PSV provided equivalent gas exchange to assist-control ventilation but with lower WOB and $\dot{V}_{O_2}$. There are no reports in the literature comparing WOB between volume control and pressure control ventilation (PCV). Because of the similarity in the pattern of inspiratory flow between PSV and PCV, a decrease in WOB during PCV can be reasonably expected. Although both of these modes suffer the drawback of inconsistent V_T delivery, PCV has the advantage of providing a minimum rate, as well as providing control over either the t_I or I:E for greater control of V_T.

The use of sedation and/or paralysis may also help decrease WOB_{pt} that results from pain, agitation, and combativeness. However, paralysis can result in retained secretions, bowel immotility, and other physiologic sequelae. Additionally, use of paralysis has been associated with prolonged weakness for days to months after the paralysis has been discontinued, may decrease the ability to wean from mechanical ventilation, and may increase ICU stay.[21]

Our study examined the effects of a new ventilatory technique on patient WOB. The ventilator modification allows the initial phase of inspiration to have the highest flow, similar to a pressure support breath, while maintaining a relatively constant V_T. It is important to point out that we took care in attempting to match $\dot{V}_{set}$ to the patient's demand for flow in the traditional AMV mode. In spite of this attempt, patients consistently selected a higher $\dot{V}$ than was thought to be ideal at the time the settings were chosen. Not only was this difference statistically significant, but it is clinically important. Should either the practitioner fail to be diligent in setting $\dot{V}$

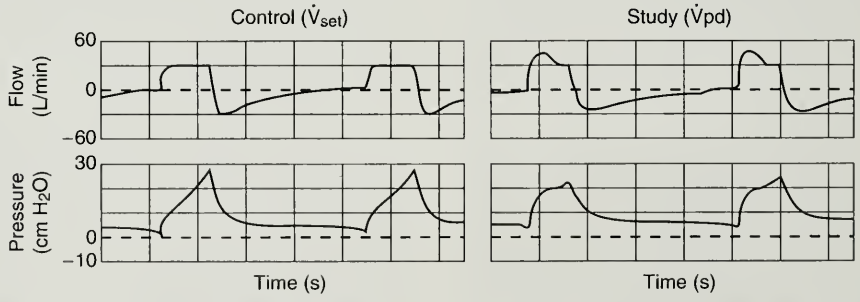

Fig. 1 Flow (top) and pressure (bottom) waveforms for Patient 13 during the control (left) and study (right) periods. Note that during the control period, the measured inspiratory flow remained constant ($\dot{V}_{set}$) at approximately 30 L/min, although the machine was set at 40 L/min. During the study period, the patient was able to determine the inspiratory flow. ($\dot{V}_{pd}$). During the early part of inspiration, flow exceeded $\dot{V}_{set}$, but decreased during late inspiration to the level of $\dot{V}_{set}$ while the remainder of the tidal volume was delivered.

or the patient's needs change quickly, the increase in the work-of-breathing indices will be more dramatic. We believe that the ability of the ventilator to match patient demand on a breath-to-breath basis may help prevent excessive WOB seen during exertion, movement, and procedures. Several of the studies already cited involved adjusting the $\dot{V}_{set}$ through a wide range.[3,4,19] We chose to match the patient's inspiratory demand with the set flow and then compare the two modes. It is probable that the magnitude of difference seen between our groups would have been much greater had we used a lower $\dot{V}_{set}$ to create a certain degree of ventilator dyssynchrony.

This study indicates that PTP, which is a measure of the mechanical work of breathing and the isometric forces, was reduced. P_{es}, another marker of patient work of breathing, was also reduced. Although $\dot{V}_E$ and f were statistically significantly different between control and study periods, the difference was not clinically important.

Reducing patient WOB is an integral component of successful mechanical ventilation. Choosing ventilator settings and a mode that enhances patient-ventilator synchrony while reducing WOB should be routine practice for bedside caregivers. The results of previous research demonstrates that set inspiratory flow is a key determinant of patient WOB. The use of the VAPS mode on the Bird 8400-ST appears to help reduce the WOB under quasi-steady state conditions. We believe these improvements may be of even greater importance during periods of stress or exertion.

The plethora of ventilator techniques—PSV, PCV, VAPS, and pressure augmentation may all be useful in reducing WOB_{pt}. However, it remains the responsibility of clinicians to assess patient-ventilator interaction, determine the cause of dyssynchrony, and apply the appropriate solutions.

PRODUCT SOURCES

Ventilator & Accessories:
Ventilator, Model 8400-ST
Wick Humidifier Model KBD 401
Nondisposable breathing circuit
Bird Products Corp, Palm Springs CA

Measuring Equipment & Accessories:
SmartCath Esophageal Balloon Catheter
VarFlex Flow Transducer
Calibrated Respiratory Mechanics Monitor Model CP-100
Bicore Monitoring Systems, Irvine CA
Pneumoguard 1200 Auxiliary Monitor, Novametrix Medical Systems, Wallingford CT

Statistical Analysis Software:
Systat v. 5.0, SPSS Inc, Chicago IL

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Measurement of Airway Occlusion Pressure ($P_{0.1}$) by a Semi-Automated Bedside System

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Recently, several studies have shown that airway occlusion pressure ($P_{0.1}$) generated in the first 100 milliseconds of inspiration is a useful noninvasive test to predict the outcome of patients weaning from mechanical ventilation. However, to perform the test a breathing circuit, transducers, and recording equipment must be assembled, and data to determine $P_{0.1}$ must be manually extracted. In this study, we compared $P_{0.1}$ measurements taken simultaneously by a computerized bedside respiratory cart and a transducer-oscillographic recording system. Studies were performed in 6 healthy subjects. Airway pressure was simultaneously measured by the semi-automated system and a differential pressure transducer, digitized to a computer-based data-acquisition system. Subjects were encouraged to vary breathing pattern to produce a wide range of $P_{0.1}$ values. The occlusion pressure waveforms collected were independently reviewed and $P_{0.1}$ visually determined. Seventy-four occlusions were conducted with $P_{0.1}$ values ranging from 0.3 to 26.2 cm H_2O. The semi-automated system rejected 21 occlusions (28%) as being unacceptable. However, the majority of these rejections were due to the operator's arming the occlusion device prematurely. High correlation ($r = 0.999$) was observed between the $P_{0.1}$ determined by the semi-automated system and the manual assessment (slope = 0.98, intercept = –0.18), indicating that the semi-automated system that we studied made accurate determinations of $P_{0.1}$ over a wide range of breathing patterns. Studies are needed to determine their applicability in measuring $P_{0.1}$ in the ventilated ICU patient. [Respir Care 1995;40(7):722-727]

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Introduction

Airway occlusion pressure ($P_{0.1}$) is the pressure generated during isometric contraction of the respiratory muscles in the first 100 milliseconds (ms) of inspiration at functional residual capacity (FRC) against an occluded airway.[1] In normal subjects, $P_{0.1}$ bears a constant relationship to other measures of central neuromuscular output (eg, diaphragmatic electromyographic activity and mean inspiratory flowrate) and has been shown to be a reproducible index of central respiratory drive.[2,3] Recently, $P_{0.1}$ has been reported to be a useful adjunct to other bedside respiratory measurements in predicting weaning success .[4,5] The predictive value of this index in assessing weaning outcome coupled with its noninvasive nature makes $P_{0.1}$ measurement a potentially attractive bedside tool.

Bedside measurement of $P_{0.1}$, however, can be cumbersome. $P_{0.1}$ measurement requires assembling a breathing circuit, an occlusion valve, pressure transducers, and recording equipment. Then, following collection of measured variables, data must be manually extracted to determine $P_{0.1}$, further increasing the time and complexity of the procedure. Finally, when performing measurements of $P_{0.1}$, the stability of the patient's breathing pattern must be assessed throughout the study in order to ensure the reproducibility of $P_{0.1}$ measurements.

Recently, semi-automated computerized bedside systems have been introduced, with the potential to simplify the $P_{0.1}$ test and easily provide noninvasive measurement of respiratory drive in the ventilated patient. The Adult Lung Function Analyzer* (ALFA) is such a computerized portable unit capable of assessing bedside pulmonary mechanics including $P_{0.1}$. This system measures $P_{0.1}$ in a semi-automated fashion that includes: (1) occlusion valve control; (2) airway pressure sampling; (3) $P_{0.1}$ determination; and (4) an evaluation of the measurement's technical quality. ALFA also provides real-time graphic display of airflow, tidal volume, and airway pressure and the monitoring of breathing pattern throughout the study period.

Our goal in this study was to evaluate the accuracy and reliability of $P_{0.1}$ measurements obtained by a semi-automated computerized monitor in order to determine its applicability for bedside measurements in the ventilated ICU patient. To accomplish this goal, we compared $P_{0.1}$ measurements made over a wide range of breathing patterns in normal subjects using a semi-automated monitor and simultaneous measurements made using a conventional manual-transducer oscillographic recording system.

Methods

Subjects

All measurements were obtained in 6 healthy subjects who gave their voluntary and informed consent to participate in this study. The protocol was approved by our institutional review board for research on human subjects.

Apparatus

Measurements of $P_{0.1}$ were performed in all subjects while they breathed spontaneously through a respiratory circuit consisting of a mouthpiece, noseclip, pneumotachograph, nonrebreathing valve and an inspiratory-port pneumatic occlusion valve. A Y-connector on the mouthpiece pressure port allowed the simultaneous measurement of airway (mouthpiece) pressure (P_m) by the ALFA system and an independent differential pressure transducer (range ± 50 cm H_2O). The Validyne transducer output was digitized (1000-Hz sampling rate), stored and analyzed by a computer-based data-acquisition system. The ALFA pressure transducer and differential transducer were calibrated simultaneously over a range of ± 30 cm H_2O against a water manometer, prior to the study of each subject.

Study Protocol

During the test, all subjects were seated upright, with the breathing circuit independently supported and a comfortable but secure seal maintained around the mouthpiece. Subjects were coached to achieve a wide range of respiratory efforts (ie, hypo- to hyperventilation) in order to produce a wide range of inspiratory occlusion pressures. At each level of respiratory effort, a stable breathing pattern was maintained for approximately 1 minute. A minimum of 10 occlusions was applied to each subject. Each occlusion was separated by at least 30 seconds.

Evaluation of the Manual System

Collection, analysis, and reporting of $P_{0.1}$ measurements by the manual system was a three-step procedure: (1) calibration; (2) data collection with manual keyboard entry of event markers; and (3) comments and report generation. Calibration of the P_m and flow transducers was recorded prior to each test. Because the occlusion valve was operated by the ALFA system, the only operator function associated with the data-acquisition system during the test was to initiate or stop data acquisition simultaneously with the ALFA system operation. After all testing was complete, the occlusion-pressure waveforms collected on the computerized data-acquisition system were independently reviewed by two of the investigators in a blinded fashion. The onset of the inspiratory negative pressure deflection was determined by locating the last P_m zero-crossing point prior to the negative P_m deflection associated with the occlusion. The $P_{0.1}$ was measured by scrolling a cursor 100 ms beyond this datum and recording the P_m value under the cursor. The criteria for accepting a manually analyzed $P_{0.1}$ were twofold. First, the occluded breath had to be preceded by two or more breaths of comparable volume and timing (subjectively assessed from the air-flow waveform). Second, the occluded breath had to have a well-defined zero-pressure crossing and artifact-free negative pressure deflection for at least 100 ms.

Evaluation of Semi-Automated System

Collection, analysis, and reporting of $P_{0.1}$ measurements by the ALFA system is a 5-step procedure: patient data entry, calibration, respiratory monitoring, data collection, and report generation. After entering patient data and calibrating the P_m and flow transducers, the operator selected the res-

*Products and suppliers are listed in the Product Sources section at the end of the text.

piratory drive assessment from a menu and entered the system into a monitoring mode. Data collection was initiated when the operator armed the occlusion valve to be automatically triggered during the next breath. The ALFA system then reported the $P_{0.1}$ measurement and any associated error codes. Measurements were deemed unacceptable by ALFA when: (1) an unstable breathing pattern was identified (eg, differences in successive tidal volumes > 30%); (2) the operator failed to allow at least 3 complete breaths prior to activating the occlusion valve (eg, insufficient data for analysis); or (3) data were incompatible with monitor mode (eg, P_m recorded during occlusion at >0 cm H_2O or a volume change during occlusion,). Graphic displays of an acceptable and unacceptable occlusion are illustrated in Figures 1A and 1B. At the completion of the test sequence, a report of the $P_{0.1}$ measurements and ventilator settings (ie, $\dot{V}_E$, V_T, f) was generated and printed.

Results

Seventy-four occlusions were performed, with simultaneous measurements of $P_{0.1}$ made by the semi-automated and the conventional manual transducer/oscillographic recording systems. Table 1 compares $P_{0.1}$ measurements obtained by both methods. During four occlusions, $P_{0.1}$ was unmeasurable by both systems due to a noisy P_m signal. Of the 70 measurable occlusions, 17 were deemed technically unacceptable by the semi-automated system, whereas only 2 of the 70 occlusions were determined to be unacceptable by the manual method. Thus, overall rejection rate of measurements was 24% by the ALFA system and 3% by the manual system.

Table 1. Summary of Airway Occlusions Performed Using Both a Manual Transducer Oscillographic System and a Semi-Automated System.

	Manual	ALFA
Total number of occlusions	74	74
Total number of 'measurable' occlusions	70	70
Number of occlusions rejected	2	17
Number of occlusions falsely rejected	0	15
False rejection rate	NA*	88%

* Not applicable – rejections were entirely subjective, though preset-criteria based.

To determine why ALFA falsely rejected 15 $P_{0.1}$ measurements (false rejection rate 15/17 = 88%), we reviewed the specific error codes assigned to these rejected breaths by the semi-automated system. Of 15 false rejections by ALFA, 13 were due to insufficient preocclusion data. ALFA found the remaining 2 rejected occlusions unacceptable due to an unstable breathing pattern (ie, excessive V_T differences) during the 3 breaths prior to the occlusion. Thus, the majority of false rejections by the semi-automated system was due to the operator's failure to allow at least 3 complete breaths before arming the occlusion valve. It should be noted that breathing pattern stability was not quantitatively assessed by the manual system, but was left to the subjective discretion of the operator.

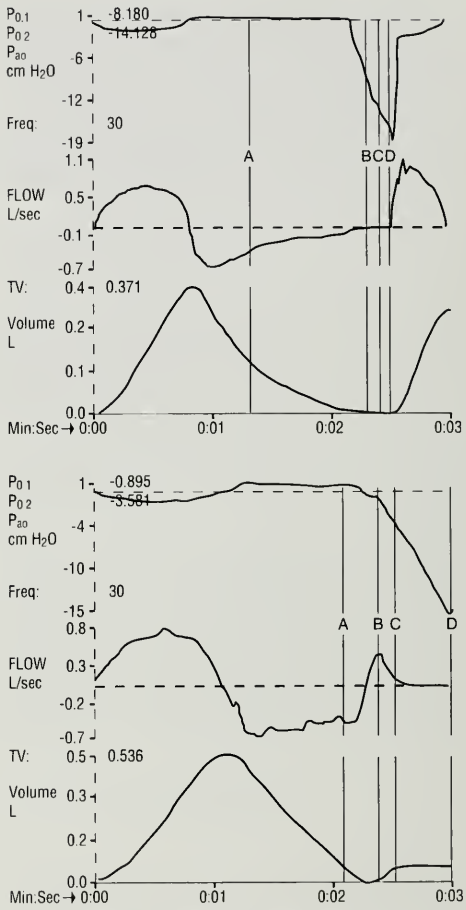

Fig. 1(A). Scalar plot of measured and calculated respiratory variables during a representative, technically acceptable airway occlusion and $P_{0.1}$ measurement. Freq (average respiratory frequency) and TV (tidal volume) were calculated from the breaths preceding the occlusion. (B) Scalar plot of measured and calculated respiratory parameters during a representative technically unacceptable occlusion and $P_{0.1}$ measurement. Note presence of flow and volume changes during a period of occlusion. *A: occlusion valve closed; B: $P_{0.1}$; C: $P_{0.2}$; D: occlusion valve open.*

Values for $P_{0.1}$ ranged from 0.3 to 26.2 cm H_2O. Figure 2 shows the linear regression of $P_{0.1}$ simultaneously measured

by the semi-automated and manual methods. $P_{0.1}$ values reported but not accepted by ALFA (n = 70) in this analysis did not significantly change the relationship (r = 0.987, slope = 0.999, intercept = –0.30) between these two measurement techniques (Fig. 2A). Linear regression of $P_{0.1}$ measurements in the range reported in studies predicting weaning outcome (ie, 0-6 cm H_2O) also showed close correlation (r = 0.96) between the semi-automated and manual methods (Fig. 2B).

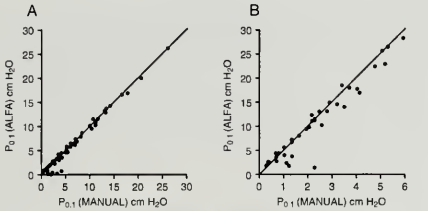

Fig. 2. Panel A shows a linear regression of all $P_{0.1}$ values (n = 70 including 17 rejected by the semi-automated system) simultaneously measured by the semi-automated and manual oscillographic recording methods (r = 0.99). Panel B shows linear regression of all technically acceptable $P_{0.1}$ values (n = 38) within the clinical range reported for predicting weaning outcome, simultaneously measured by both systems (r = 0.96).

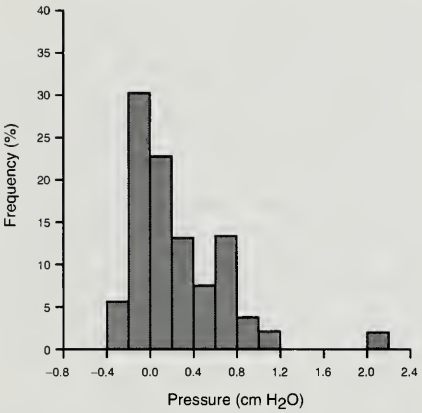

Fig. 3. Histogram of differences between the semi-automated and manually determined $P_{0.1}$ measurements for the 53 technically acceptable $P_{0.1}$ measurements. The manually determined values of $P_{0.1}$ tended to be slightly greater than the semi-automated derived values with a shift in the histogram frequency curve to the left.

Although the slope and intercept of the relationship between the ALFA and manually derived $P_{0.1}$ values approach identity, a further review of the data was performed to assess the magnitude of the measurement error between the two systems. Figure 3 shows a histogram analyzing the differences between the ALFA and manually measured values of $P_{0.1}$ generated for the 53 technically acceptable occlusions by the semi-automated system. These differences were derived by subtracting the $P_{0.1}$ value measured by the semi-automated system from the $P_{0.1}$ value measured simultaneously by the manual system. Differences between these simultaneous $P_{0.1}$ measures ranged from –0.4 through 2.2 cm H_2O, with greater than 90% of the differences narrowly clustered between –0.4 to 0.8 cm H_2O. Overall, the $P_{0.1}$ values measured by the semi-automated system were slightly smaller ($p < 0.001$) in magnitude than the manually derived values.

Discussion

Our data show that $P_{0.1}$ measurements made by a semi-automated system provide accurate measurements in normal subjects over a wide range of breathing patterns, compared to a conventional manual oscillographic recording system. Measurements of $P_{0.1}$ simultaneously obtained by both of these methods were highly correlated regardless of the technical quality of the individual occlusion. Although the manual method provided the same value for $P_{0.1}$ in the majority of measurements, no information was provided to assess the reliability of the measured value. That assessment was purely subjective. The semi-automated system provided not only an automatic evaluation of the technical quality of the occlusion, but also provided continuous graphic monitoring to determine the reliability with which the $P_{0.1}$ measurement was made. These data suggest that semi-automated systems may provide accurate and reliable measurements of $P_{0.1}$ over a wide range of breathing patterns.

$P_{0.1}$ has recently been described as a useful clinical parameter to measure when assessing a patient's ability to be weaned from mechanical ventilation. Sassoon et al[4] studied 12 patients in respiratory failure treated with mechanical ventilation and found that those patients with $P_{0.1}$ values higher than 6 cm H_2O measured within 5 minutes of beginning a spontaneous breathing trial were unable to be successfully weaned from mechanical ventilation. Murciano et al[3] found $P_{0.1}$ to be significantly elevated (ie, > 7 cm H_2O) in 16 patients with COPD and respiratory failure who required mechanical ventilation. Those patients who were able to be successfully weaned from mechanical ventilation showed a progressive decline in $P_{0.1}$ over 3 days that correlated with a reduction in the electromyographic evidence of diaphragm fatigue. Patients who showed electromyographic evidence of persistent diaphragm fatigue and sustained elevations in $P_{0.1}$ values failed repeated attempts at weaning and extubation.

Montgomery et al[6] measured $P_{0.1}$ before and after brief CO_2-rebreathing challenge in 11 patients in respiratory failure who received mechanical ventilation for a variety of medical and surgical disorders. Those patients who showed a significant increase in $P_{0.1}$ during hypercapnic challenges were successfully weaned from mechanical ventilation. The authors concluded that the ratio of $P_{0.1}$ measurements made before to those

made after hypercapnic challenge was a sensitive and specific means by which the success or failure of weaning from mechanical ventilation could be predicted.

It should be noted, however, that none of these studies evaluated the reliability of $P_{0.1}$ as a weaning parameter in a prospective and randomized, controlled fashion. Nonetheless, all studies suggested that the $P_{0.1}$ is a potentially useful clinical measurement to predict weaning outcome. Moreover, a simple and reliable bedside method to measure $P_{0.1}$ would make such a measurement accessible to the clinician in everyday practice.

Bedside measurement of $P_{0.1}$ by conventional methodology, as performed in the previously mentioned studies, may be somewhat cumbersome and is fraught with potential inaccuracies that may easily go undetected. Conventional methods of measuring $P_{0.1}$ requires assembling of a breathing circuit, transducers, and recording equipment, such as the manual oscillographic recording system that we used. Such equipment is not designed to be portable and requires careful calibration against a reference standard prior to use. Patients must be connected in line with a breathing circuit that minimizes dead space, and a stable breathing pattern should be ensured prior to $P_{0.1}$ measurement. Arming of the pneumatic occlusion valve should go unnoticed by the patient to avoid the conscious knowledge of an impending inspiratory occlusion that may modify inspiratory effort and influence $P_{0.1}$ measurement. In addition, the operator must be able to open the occlusion valve rapidly, so as not to obstruct the ensuing inspiration and to minimize discomfort to the patient, especially those patients who are moderately ill and weaning from mechanical ventilation. Depending upon the manner in which these valves are armed and disarmed, a certain amount of skill, coordination, and training is required of the operator.

The semi-automated system we used addresses many of these issues by simplifying the bedside measurement of $P_{0.1}$ and providing the means to assess the accuracy and reliability of $P_{0.1}$ measurements. This unit is self-contained, portable and simple to calibrate. In a semi-automated fashion, it provides occlusion valve control, airway pressure sampling, measurement of $P_{0.1}$, and an evaluation of the measurement's technical quality. Moreover, continuous graphic monitoring of lung mechanics allows one to assess stability of the breathing pattern to ensure accurate $P_{0.1}$ measurement. Occlusion valves are closed and opened quietly and rapidly and are, thereby, minimally occluded only for the time necessary to obtain $P_{0.1}$. In this way, patient discomfort is minimized and significant delays in making the measurement are avoided. Finally, because of its semi-automatic features, there is minimal operator skill and training required to measure $P_{0.1}$.

In our study, the semi-automated system rejected approximately 25% of all $P_{0.1}$ measurements as technically inadequate. Visual review of these rejected occlusions as measured simultaneously by the manual system showed that most of these values were acceptable. A majority of these rejections (13/15) were based on insufficient preocclusion data upon which the stability of the breathing pattern is based (ie, the operator failed to allow at least 3 breaths to precede an occlusion). This is a correctable error that should diminish as operator experience and skill increase. The remaining false rejections were due to the semi-automated system's detecting excessive tidal volume differences in the breaths preceding the occlusions and, thus, concluding that an unstable breathing pattern existed.

In spite of the high overall rejection rate, occlusions may be repeated every 30 seconds to obtain a reliable set of values in a timely manner. A manual override feature is available that allows the operator to accept values that the semi-automated system may have rejected by its predefined set of criteria. By using the semi-automated system, occlusions were performed quietly, efficiently and repeatedly without alerting the subject to the arming of the valve and the impending inspiratory occlusion.

Recently, others have reported bedside methods of measuring $P_{0.1}$ in ventilated patients. Brenner et al[7] performed occlusions via a servo ventilator using digitalized signals and computer software to automatically determine $P_{0.1}$. In nonventilated normal subjects and ventilated ICU patients, they found that this system produced accurate and automatic determinations of $P_{0.1}$. In comparison to performing $P_{0.1}$ measurements by the system outlined by these investigators, the semi-automated system that we studied has several advantages. First, ALFA automatically evaluates the technical reliability of a given occlusion and provides data for manual confirmation. Furthermore, whereas newer ventilators may be adapted to measure $P_{0.1}$, earlier models may not be, thus, limiting the availability of the $P_{0.1}$ measurement only to hospitals that have newer mechanical ventilators. Probably the greatest advantage of the semi-automated unit is the capability afforded the clinician to ensure stability of breathing. This feature ensures the accuracy and reliability of the $P_{0.1}$ measurements. This is especially important in the subgroup of patients for whom bedside determination of $P_{0.1}$ might be most important (ie, patients with obstructive lung disease being weaned from mechanical ventilation).

In summary our data show that a semi-automated bedside respiratory mechanics monitor measures $P_{0.1}$ in a reliable and accurate manner in spite of a wide range of breathing patterns. The application of semi-automated systems to measure $P_{0.1}$ in critically ill patients being weaned from mechanical ventilation needs to be investigated.

PRODUCT SOURCES

Lung Function Analyzer:
ALFA, Pulmonary Technology Industry Inc, Hatfield PA

Pneumotachograph:
Fleisch #2, Gould Electronics, Cleveland OH

Nonrebreathing Valve:
#2600, Hans Rudolph, Kansas City MO

Pneumatic Occlusion Valve:
Medical Associated Services, Hatfield PA

Pressure Transducer:
Model MP50, Validyne Engineering Corp, Northridge CA

Data Acquisition System:
Codas System, Dataq Instruments Inc, Akron OH

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Accuracy of Laminated Disposable Pulse-Oximeter Sensors

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BACKGROUND: In our hospital, widespread utilization and clinical importance of arterial oxyhemoglobin saturation monitoring by pulse oximetry (S_{pO_2}) generated a growing cost-center in a time of fiscal restraint. We hypothesized that lamination of a pulse-oximeter sensor and insertion into a disposable, protective shield allowing reusability would not alter sensor response time or relative accuracy. MATERIALS AND METHODS: Six healthy adult men volunteered to be studied during hypoxemia (induced by spontaneous ventilation of a 8-10% oxygen concentration in helium by face mask) to an $S_{pO_2} \leq 70\%$. The S_{pO_2} was monitored by 2 Nellcor N-200 pulse oximeters with D25II sensors and an Ohmeda 3700 pulse oximeter with the OxyTip sensors. Single desaturations, repeated twice in 5 subjects, and 2 desaturations, repeated 5 times in 1 subject, were evaluated. After the initial desaturation and recovery, 1 D25II sensor was laminated, inserted into a protective sheath, reattached and followed by repeat desaturation. S_{pO_2} readings from each oximeter, during both desaturations sets were compared. Clinical importance was subjectively determined by comparison to published S_{pO_2} bias data. RESULTS: 907 data sets were collected. In 447 S_{pO_2} comparisons in 5 individuals (range, 62-100%), 1 laminated sensor was 0.8 percentage points lower and one sensor was 1.3 percentage points higher ($p < 0.05$) than controls compared with prelamination differences. No difference was clinically important. In individual repeated desaturations (range, 52-100%) following lamination, one sensor was 2.9 percentage points and one sensor was 2.6 percentage points higher. During repeat desaturations, differences between the OxyTip and the D25II control sensor were 1.3 percentage points lower and 1.8 percentage points higher than the initial desaturation (all $p < 0.001$). Response times were not altered. CONCLUSIONS: Lamination and protective shield applied to disposable sensors did not alter S_{pO_2} measurements or response times to incremental reductions in S_{pO_2} in a clinically important manner. [Respir Care 1995;40(7):728-733]

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Introduction

During recent decades, noninvasive measurement of arterial oxyhemoglobin saturation by pulse oximetry (S_{pO_2}) has been considered one of the most important monitoring advances in the care of anesthetized and seriously ill patients, and its use is the accepted standard of care intraoperatively.[1] However, along with its perceived clinical importance for quality patient care and widespread applicability, a cost-center has developed in which disposable pulse-oximeter sensors have become a considerable expense for both operating rooms and respiratory care departments. Many institutions utilize disposable sensors that are designated "for single-patient use." As an example of this widespread use and expense, we have estimated that disposable sensors may cost our own 500-bed institution approximately $200,000 during a single fiscal year (1995).

Attempts at fiscal restraint have led to reuse of disposable sensors, using methods that are often cumbersome and unreliable compared to the single-use practice recommended by the manufacturers. One company has developed a process whereby the sensors are laminated, allowing cleansing and reuse, and inserted into an inexpensive, disposable probe shield.*

The accuracy of pulse oximeters for determining S_{pO_2} has been determined by comparisons to in-vitro measurements done on arterial blood-gas samples by CO-oximeters (S_{aO_2}). This accuracy (1 SD) has been reported, by the manufacturer to be 2 percentage points for S_{pO_2} values of 70-100% and 3 percentage points for values below 70%.[2] We hypothesized that lamination of a pulse-oximeter sensor and insertion into a disposable shield would not alter either response time or relative accuracy when compared to clinically used, nonlaminated sensors.

Methods

After approval by our institution's Clinical Investigations Committee, 6 healthy, adult, nonsmoking men , ages 36-42 years, volunteered to be studied. The studies were divided into 2 phases. In Phase 1, to evaluate intersubject variation, comparisons of laminated and nonlaminated sensors were made during 2 hypoxemic episodes in each of 5 subjects. In Phase 2, variation within a single individual was assessed by monitoring 2 hypoxemic episodes, repeated 5 times; a total of 20 hypoxemic episodes were performed (10 sets of 2 episodes). A new set of sensors was used to monitor each set of desaturations.

Subject Monitoring

Each subject was studied supine, and 5-lead electrocardiogram and right-arm blood pressure were continuously monitored. Respiratory gases—fraction of delivered oxygen (F_{DO_2}) and nitrogen and end-tidal carbon dioxide (P_{ETCO_2})—were monitored by Raman spectrometry. S_{pO_2} was monitored on the left hand with D25II sensors, one each on the index and middle fingers, and with an Ohmeda OxyTip sensor on the ring finger.

Induction of Hypoxemia

All hypoxemic episodes were induced in the same manner. Each subject spontaneously breathed an hypoxic gas mixture (8-10% oxygen in balance helium) through a clear, tight-fitting face mask. Helium was utilized as the carrier gas because during pilot studies subjects felt less air hunger and tended to become less tachypneic during desaturations, when helium rather than nitrogen was used. Also the absence of nitrogen in the respiratory gas was used as an indicator of the absence of room-air leak into the circuit. After S_{pO_2} decreased to $\leq 70\%$, each subject then breathed a 50% helium:50% oxygen mixture for recovery. S_{pO_2} was monitored and recorded through recovery to $\geq 95\%$ S_{pO_2}. Each subject then breathed 100% oxygen for a minimum of 10 minutes before the hypoxemic episode was repeated.

Protocol

The initial hypoxemic episode was measured by 2 D25II and 1 OxyTip sensors. During the 10 minutes prior to the second hypoxemic episode, one D25II sensor was removed, laminated, placed in a probe shield, and reattached to the same digit from which it had been removed. The other two sensors were not removed or changed in any way. During Phase 1, induced hypoxemia and recovery were completed twice on 5 individuals and later, during Phase 2, were followed by 5 pairs of hypoxemic episodes in a single subject. During desaturation and recovery, S_{pO_2} was recorded every 10 seconds for comparison. Response times—time needed to achieve a 5 percentage-point reduction in S_{pO_2} (ie, 100-95, 95-90, 90-85, 85-80, 80-75, 75-70)—were also recorded.

Data Analysis

S_{pO_2} values and response times to 5 percentage-point reductions in S_{pO_2} were compared for each of the 2 D25II sensors and the OxyTip sensors during the controlled desaturations, recovery, and after probe-shield and laminate application. For each desaturation episode, S_{pO_2} values were recorded every 10 seconds from all 3 probes. The differences in S_{pO_2} between the control (unchanged D25II) and the test sensor (laminated D25II) were calculated, as was the differences between control and the OxyTip sensor, both before and after lamination of the test sensor. The mean and SD of the differences (bias and limits of agreement) between data pairs were calculated.[3] Differences between sensor pairs and response times for each 5 percentage-point reduction in S_{pO_2} before and after lamination were compared by analysis of variance (ANOVA). Pairs from significant ANOVA were compared with Scheffé post-hoc analysis.[4] A p value ≤ 0.05 was considered statistically significant. Clinical importance was evaluated subjectively by comparison with published S_{pO_2} bias data.[5,6]

Results

A total of 907 S_{pO_2} pairs were compared in 6 individuals.

* Products and suppliers are identified in the Product Sources section at the end of the text.

Phase 1

447 sets of S_{pO_2} measurements recorded from standard disposable D25II sensors, laminated D25II sensors, and OxyTip sensors were made in 5 individuals. The S_{pO_2} values ranged 62-100%. The mean differences for S_{pO_2} values between sensors were within the manufacturer's error range (2 SDs, 3.9 percentage points) for the pulse oximeters. There was no difference between the D25II and OxyTip sensors before or after the D25II lamination (Fig. 1). However, of 10 S_{pO_2} values < 70% detected by the D25II probes, 7 were not detected by the OxyTip sensor, which had lost its ability to detect the pulse amplitude. The cause of this loss of pulse detection is unclear, although in all cases pulse detection spontaneously returned during reoxygenation.

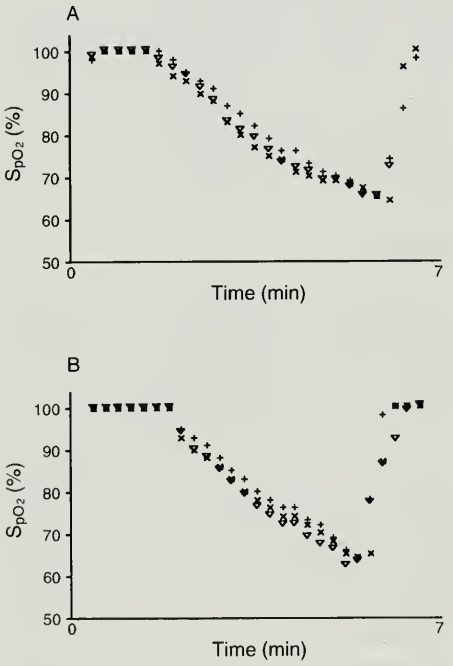

Fig. 1. A comparison of arterial oxygen saturation measurements by pulse oximeter (S_{pO_2}) in 1 subject during induction of hypoxemia by breathing an 8% oxygen: 92% helium gas mixture. The S_{pO_2}s were determined before lamination (A) by 2 Nellcor D25II sensors (+, control and ∇, test) and 1 Ohmeda OxyTip sensor (x) and after lamination (B) by the same 2 Nellcor D25II sensors, one of which had been laminated and placed in a protective shield, and the same Ohmeda OxyTip sensor. There were no clinically important differences in saturation measurements among the sensors.

The measured response times were not statistically different among sensors for measured saturation changes. There was no clinically important difference in measurements made among sensors.

There were statistically significant differences (prelamination compared with postlamination) for the 2 D25II sensors (Table 1). Following lamination, the difference between control and test sensor in one subject was 0.8 percentage points lower than prelamination ($p < 0.05$) and in one subject was 1.3 percentage points higher ($p < 0.05$). Both of these differences are within the manufacturer's published specifications of ± 3.9 percentage points (2 SDs).

Table 1. Mean ± SD Differences in S_{pO_2} between Control and Experimental Pulse Oximeter (Nellcor D25II) Sensors before and after Lamination during Desaturations in 5 Subjects.

Subject	Before	After
1	0.917 ± 0.92	2.640 ± 1.368*
2	0.977 ± 1.64	0.386 ± 2.452
3	1.739 ± 1.453	2.667 ± 2.787
4	0.409 ± 0.854	0.560 ± 0.870
5	1.658 ± 0.966	0.809 ± 2.018*

* ANOVA and Scheffé, $p < 0.05$.

Phase 2

460 comparisons of S_{pO_2} measurements from standard disposable Nellcor D25II sensors, laminated D25II sensors, and Ohmeda OxyTip sensors were made after 5 pairs of repeated desaturations in one subject. The measured S_{pO_2} values ranged 52-100%. Again the mean differences between simultaneous comparisons of S_{pO_2} were within the error range of the pulse oximeters. The response times were not statistically different among sensors. There were no clinically important differences among sensors. S_{pO_2} values below 70% were detected similarly among sensors.

Statistical analysis revealed that one laminated sensor was 2.6 percentage points and another 2.9 percentage points higher following lamination than the control sensor (both $p < 0.0001$, Table 2). Both of these differences are within the manufacturer's published specifications of ± 3.9 percentage points (2 SDs). One Ohmeda OxyTip sensor was 1.8 percentage points higher ($p < 0.001$) and one was 1.3 percentage points lower ($p < 0.001$) than the control sensor (Table 3).

Discussion

Covering disposable oximeter sensors with a protective laminate as we have described does not alter S_{pO_2} measurements or response times to reductions in S_{pO_2} to a clinically important degree and, therefore, may provide a means of cost-saving without altering the quality of patient care.

Table 2. Mean ± SD Differences in S_{pO_2} between Control and Experimental Pulse Oximeter (Nellcor D25II) Sensors before and after Lamination during Repeated Desaturations in 1 Patient.

Desaturation	Before	After
1	0.254 ± 3.474	2.955 ± 3.474*
2	−0.447 ± 1.339	−0.729 ± 1.412
3	0.974 ± 2.089	0.640 ± 1.770
4	0.195 ± 2.152	−0.103 ± 1.861
5	−1.295 ± 0.955	1.650 ± 2.413*

* ANOVA and Scheffé, $p < 0.001$.

Table 3. Mean ± SD Differences between S_{pO_2} Values Measured by Ohmeda OxyTip and Nellcor D25II Control Sensors before and after Lamination during Repeated Experimental Hypoxemic Episodes in 1 Patient.

Desaturation	Before	After
1	−3.09 ± 2.39	−2.20 ± 2.45*
2	2.77 ± 1.16	2.37 ± 0.97
3	1.22 ± 1.73	−0.10 ± 1.87*
4	1.17 ± 1.20	1.39 ± 2.06
5	−2.89 ± 2.79	−1.10 ± 2.43*

* ANOVA and Scheffé, $p < 0.05$.

Pulse Oximetry & Oximetry Sensors

The principles of pulse oximetry are well known.[7-10] Two light emitting diodes (LEDs), one for red (~ 660 nm) and one for infrared (~ 940 nm) light, emit light through tissue at frequencies as high as several hundred hertz, depending on the particular oximeter. Light absorption is measured by a photodetector positioned on the side of the tissue directly opposite the LED, whether finger, toe, or nose. Oxyhemoglobin absorbs less red light and reduced hemoglobin absorbs less infrared light. Absorption by tissues varies with the cardiac cycle—the absorption difference during diastole and systole caused primarily by arterialized blood. The relative absorption at the two light wavelengths is used to calculate S_{pO_2}.

Pulse Oximeter Accuracy

Pulse-oximeter sensors have clinically applicable accuracy in their estimation of actual arterial oxyhemoglobin saturation. The manufacturer's accuracy of the D25II and Nellcor pulse oximeter as presented in the package insert is within 2 percentage points from 70-100% S_{pO_2} (1 SD) and to within 3 percentage points from 50-69% S_{pO_2} (1 SD). This implies that if the S_{aO_2} (measured by CO-oximeter) is 90%, then the S_{pO_2} will read between 88% and 92% only 66.6% of the time and between 86% and 94% (2 SDs) approximately 95% of the time.

Accuracy of S_{pO_2} compared to S_{aO_2} with 79 data sets in 5 adult volunteers has been reported by Yelderman and New[5] to be within 5 percentage points S_{pO_2} (2 SDs) at values from 70-100% S_{pO_2}. However the percent bias (expressed as $[S_{aO_2}-S_{pO_2}]$) varies widely. In adult volunteers studied by Severinghaus and Neifeh,[6] bias varied from +9 to −13.1 percentage points. Utilizing the Nellcor N-200 with 58 data sets, bias was reported as +2.4 with an ear sensor and +4.5 with a finger sensor; the S_{pO_2} range was 40-70%. This same study reported on 60 data sets obtained by using the Ohmeda 3700 oximeter and found a bias of −2.4 for ear sensors and +9.0 for finger sensors. Other studies evaluating adult inpatients,[11,12] neonates,[13,14] intensive care unit patients,[15,16] and canines[17] have found similar variation in bias calculations.

Most comparisons of in-vivo S_{pO_2} measurements and in-vitro S_{aO_2} measurements have shown increasing bias with lower saturation values. For example, bias has been reported to be +11.2 percentage points between 50 and 60% S_{pO_2}, but only +3.88 percentage points between 70 and 75% S_{pO_2}.[18] Recently, the error of Nellcor pulse oximeters has been shown to increase at low S_{pO_2} values, with 40% of the errors being greater than 5 percentage points.[19]

Financial Implications of Reusing Disposable Sensors

Monitoring of S_{pO_2} has extended from the operating room, to intensive care units, surgical and medical step-down units, and into the office and home care setting. This widespread utilization has resulted in the development of an extensive cost-center for medical institutions. In our experience, disposable S_{pO_2} sensors may cost individual institutions from $9 to $15 each. The actual cost can vary because of bulk buying and multi-institutional buying consortia. The authors' institution is a 500-bed tertiary care center with approximately 12,000 surgeries yearly and approximately 70 intensive-care beds. Approximately 22,000 disposable S_{pO_2} probes are used annually in our institution. While the maximum number of possible reuses has not been determined, Gerber et al[20] found no alterations in sensor accuracy with 2 to 5 reuses when the lamination and protective sheath were incorporated. Because oximetry sensor function is based on LED technology, as many as 10 reuses may not be an unreasonable expectation. This would reduce the number of new sensors we require annually to 2,200. The lamination and protective sheath costs approximately $3 for each sensor. The total cost (new probe and lamination cost) would therefore decrease to $26,400. Our institution could save more than $150,000 annually, assuming 10 separate sensor uses. The potential cost-savings of reusing accurate disposable sensors may be substantial.

Potential Problems with Sensor Reuse

Disposable pulse-oximeter sensors have been reused in many different applications. Simply reapplying the same adhesive attachment can lead to unreliable sensor contact and position as well as potential obstruction of the light source and/or the

photodetector.[21] Other user modifications have included wrapping in plastic wrap[22] and attaching self-grasping connecting material to the sensor.[23] However, with any modification, sensitivity and response time must not be affected and reliably and consistency of application is necessary.

Possible bacterial contamination of reusable medical equipment is always a concern. This concern has been raised for stethoscopes[24] and blood pressure cuffs[25] and now, reusable pulse-oximetry sensors.[26] Nosocomial infections can be especially devastating to immunologically suppressed patients, such as transplant recipients, those receiving corticosteroids, and those with prolonged, serious illnesses. Although bacteria including *Staphylococcus aureus*, *Enterococcus faecalis*, and *Klebsiella oxytoca* were grown from 66% of oximeter sensors in one study,[26] the issue is adequate decontamination rather than reusability.

New product information for some disposable pulse-oximeter sensors advise "single patient use only." However, practical considerations are often applied to many single-use items. A random hospital survey in 1988 found that 58% of hospitals were reusing medical equipment designed for single use only.[27] The Centers for Disease Control and Prevention Guidelines recommend individual hospital assessment of safety, efficacy, and functional applicability of items rather than prohibiting reuse of single-use items, and clinical utility must be determined at user sites.[28,29]

Limitations of the Present Study

We compared two pulse-oximeter sensors with one that had undergone a lamination process and was inserted into a disposable, protective sheath. No clinically significant difference was found in the response times or S_{pO_2} measurements made in volunteers with S_{pO_2} values ranging 59-100%. The study evaluated the relative accuracy of the sensors before and after a lamination process. We did not compare these sensors to oxyhemoglobin saturation measurements made on arterial blood by in-vitro CO-oximetry, which is considered the reference standard. The purpose of this study was to compare the laminated sensors to currently commercially available disposable sensors, and the absence of S_{aO_2} data does not alter the lack of clinically important differences shown by our data. These studies were performed over a short time period. Each study—from normal S_{pO_2} to desaturation to full recovery—generally took less than 10 minutes. This allowed better comparison of response time, allowed a wide range of S_{pO_2} to be measured, and was safe for the volunteers. However, there is no reason to think that long-term monitoring would alter the sensitivity and response time of any sensor.

In Conclusion

Covering disposable oxygen-saturation sensors with a protective laminate did not alter S_{pO_2} measurements or response times to incremental reductions in F_{DO_2} to a clinically important degree. This process may provide a means of cost-savings for S_{pO_2} monitoring without altering the quality of patient care.

PRODUCT SOURCES

Probe Shield/Laminate:
Medical Taping Systems, Glenmore PA

Electrocardiogram:
Model 78534C, Hewlett Packard, Mountainview CA

Blood Pressure Monitor:
Dinamap, Critikon Inc, Tampa FL

Raman Spectrometry:
Rascal II, Ohmeda Inc, Louisville CO

Pulse Oximeters:
Model N-200, Nellcor Inc, Hayward CA
Model 3700, Ohmeda Inc, Louisville CO

Oximeter Sensors:
D25II, Nellcor Inc, Hayward CA
OxyTip, Ohmeda Inc, Louisville CO

Statistical Analysis Software:
StatView, Abacus Concepts Inc, Berkley CA

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Mechanical Ventilation in Duchenne's Disease: A Case Report

Horacio Abbona PhT RT and Ricardo Navarro MD

We present a case of successful long-term external mechanical ventilation of a patient with Duchenne's disease, using a device of our own design. After comparison with known and well-established methods of external mechanical ventilation, we conclude that the primary ventilatory requirements of neuromuscular patients like ours can be met by this simple, low-cost device. Although it may not be the ventilator of choice, it may still be useful in particular situations where other ventilatory modes are either unsatisfactory to the patient or are unavailable. Our patient, a 20-year-old man was ventilated at home for more than 4 years using this device. He eventually succumbed to overwhelming left-ventricular failure. [Respir Care 1995;40(7):734-736.]

Introduction

Duchenne's disease is a hereditary muscular dystrophy of relatively rapid evolution. Death occurs most commonly between adolescence and early adulthood usually due to respiratory insufficiency associated with pulmonary infection. Patients are commonly confined to a wheelchair, and, late in the course of the disease as a result of respiratory muscle compromise, may require assisted ventilation.

We present a patient with Duchenne's muscular dystrophy in whom successful long-term ventilatory support was provided at home, using a device of our own design.

Case Presentation

A 20-year-old man known to have Duchenne's disease was admitted to the hospital because of severe dyspnea. Duchenne's muscular dystrophy was diagnosed at the age of 5 years. At 10 years of age, he was confined to a wheelchair, and muscular weakness continued to progress. One year before his admission to the hospital, he began to have shortness of breath that was also worsening. Physical examination showed a severely dyspneic, cyanotic man with extreme generalized muscular atrophy and paralysis of arms and legs. Spontaneous ventilation was minimal—there was some accessory respiratory muscle activity but no evidence of diaphragmatic activity. During the 10 hours preceding his admission, he had been receiving assisted ventilation by manual anteroposterior chest compression, administered by family members.

Chest radiograph showed clear lung fields and a marked reduction of the anteroposterior diameter of the chest. Pulmonary function tests could not be performed—only a vital capacity of 500 mL was obtained. Arterial blood-gas analysis done while the patient breathed room air showed pH 7.37, P_{aCO_2} 66 torr, P_{aO_2} 47 torr, total CO_2 40 mEq/L, base excess 8.6 mEq/L.

Tracheostomy with conventional positive-pressure mechanical ventilation was rejected by both the patient and his relatives. Therefore, because other known systems of noninvasive mechanical ventilation were not available, alternative systems were tried.

First, we attempted to ventilate this patient by applying a 1.5-L plastic bag that was connected to a volume ventilator over his abdomen. The bag was covered with a semirigid wrap, forcing the abdomen to be compressed during the ventilator's inspiratory phase. The subsequent passive abdominal decompression would theoretically cause pulmonary insufflation, but it was insufficient for this patient. Next, the same bag was applied over the anterior chest wall, and covered with a plate of wood that had been fixed to a strong fabric covering the patient's back. Insufflation of the bag by the ventilator com-

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pressed the anterior chest wall, and the reflex thoracic decompression resulted in adequate tidal volume exchange.

The patient adapted rapidly. Tidal volume increased from 200 to 500 mL and P_{Imax} from −15 to −30 cm H_2O. Initial blood-gas values were pH 7.39, P_{aCO_2} 71 torr, P_{aO_2} 34 torr, and after 15 minutes of mechanical ventilation were: pH 7.64, P_{aCO_2} 34 torr, P_{aO_2} 79 torr. The patient complained of dizziness, which was probably related to hypocapnia.

After some months, the ventilator was replaced by another portable electric device that incorporated a piston to inflate the bag (Fig. 1). The patient used external mechanical ventilation for more than 4 years (from March, 1985 to August, 1989). During that time, he had several episodes of respiratory infections and had difficulty clearing his bronchial secretions. From time to time, the patient required daily aspirations with the fiberoptic bronchoscope for periods of several weeks. These procedures were performed in the hospital on an outpatient basis. This patient was not readmitted to the hospital and was able to complete his university studies.

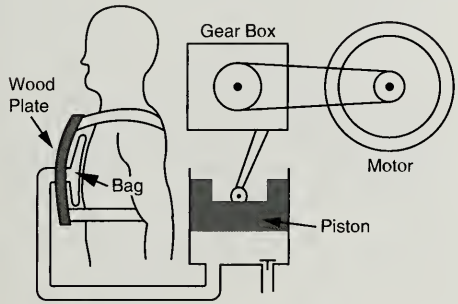

Fig. 1. Diagram of chest compression device. The device also includes a low-pressure circuit alarm and a pressure-relief safety valve set at 100 cm H_2O. The degree of chest compression is regulated by tightening the wood plate.

Fifty-three months after mechanical ventilation was initiated, he was admitted to the hospital because of sudden-onset dyspnea, cough, and copious frothy sputum. Acute pulmonary edema was diagnosed and treatment initiated with morphine and furosemide. An echocardiogram showed a dilated and hypertrophied left ventricle. He died on Hospital-Day 3 after he developed a new episode of dyspnea and did not respond to oxygen, morphine, or diuretics.

Discussion

Duchenne's muscular dystrophy is a lethal X-linked, recessive genetic muscle disease that has been found in patients from every part of the world. It is incident on 1 in 3,300 male births. Usually, it progresses rapidly, with death occurring during adolescence. Only occasionally does a patient survive beyond the age of 25 years. Respiratory muscle compromise appears late in the course of the disease, with loss of diaphragmatic function as a final event. The cause of death is commonly respiratory insufficiency with hypoxemia and hypercapnia, although, in 10% of cases, cardiac decompensation is also a main or contributing factor resulting from cardiac muscle-fiber loss and replacement fibrosis.[1]

The appearance of significant hypoxemia and hypercapnia indicates that these patients need some form of mechanical ventilation, whether invasive or not or whether nocturnal or continuous.

In choosing a system of domiciliary mechanical ventilation, three kinds of devices should be considered, each of them with its own advantages and disadvantages.

Positive-Pressure Devices

These have the capability of providing almost any requirement of ventilation. Size and portability are, in recent models, acceptable. The main drawbacks are high cost and the need for tracheostomy, which poses its own risks of complications, and the inability of the patient to speak, which adds another limitation to a severely disabled patient.[2]

Noninvasive positive-pressure ventilation (NIPPV) was initially described in 1969[3] and achieved widespread use after 1987.[4-8] However, this option was not available to us at the time our patient required treatment. NIPPV may be administered by volume- or pressure-cycled ventilators via a facial or nasal mask or a mouthpiece. NIPPV has become the method of choice for domiciliary ventilatory support because of its effectiveness and because patients prefer it. Advantages of NIPPV include improved pulmonary ventilation, fewer hypercapnia-related symptoms, sustained improvement in arterial blood-gas values, improved quality of sleep, and decreased rate of hospitalization.[4,9,10] Nevertheless, several challenges remain for NIPPV—air leaks, facial skin lesions, and gastric distention.[3]

Negative-Pressure Devices

Although they do not attain the performance characteristics of positive pressure devices, they do avoid the need for tracheostomy. Size and portability are less than desirable; high cost and the possibility of causing inspiratory collapse of upper airways markedly reduce their usefulness.[11]

Abdominal Compression Devices

These operate based on the principle of passive inspiration. After an expiration obtained by upward displacement of the diaphragm by abdominal compression, inspirations are usually acceptable in size. These devices are portable, and there is no need for tracheostomy.[12] Nevertheless, their use is lim-

ited because the patient must remain in a sitting or reclined position, and these devices cannot be applied to obese or very thin patients.[13]

In Summary

With our case, we presented the usual difficulties in the selection of a permanent system for mechanical ventilation: (1) patient's acceptance, (2) limitations by his physical characteristics, and (3) risk of morbidity. Cost limitations and availability were the most difficult challenges for us.

Although there have been reports of good results using abdominal compression devices,[14] we failed to obtain adequate ventilation in our patient. This was probably related to obtaining enough displacement of the diaphragm because of small abdominal mass—a well-known problem with such devices.[13]

We conclude that although IPPV (invasive or noninvasive) and negative-pressure ventilation are presently the most desirable devices for long-term home ventilation, chest-compression systems might still play a role in particular situations when, for whatever reason, more effective methods are not available or are refused by the patient.

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Pulmonary Vascular Control in Hypoplastic Left-Heart Syndrome: Hypoxic- and Hypercarbic-Gas Therapy

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Various mixtures of inhaled gases have been used clinically to affect pulmonary vasomotor tone for infants with certain congenital heart defects such as hypoplastic left-heart syndrome (HLHS). Our ability to manipulate the concentrations of oxygen and carbon dioxide may be the chief determinant of survival. I report a case of an infant with HLHS who, following the Stage-1 Norwood Procedure, required treatment with $F_{DO_2} < 0.21$ and hypercarbia (15 torr) to maintain a normal ratio of pulmonary-to-systemic vascular resistance. [Respir Care 1995;40(7):737-742.]

Introduction

The use of inhaled gases to modify pulmonary vascular resistance (PVR) has been practiced for many years.[1-3] Recent studies with agents such as nitric oxide have demonstrated their important role in maintaining pulmonary vascular tone. Because oxygen is a potent pulmonary vasodilator[4,5] and carbon dioxide is an equally potent pulmonary vasoconstrictor,[6,7] the calculated manipulations of these common gases has a role in the management of the patient with hypoplastic left heart syndrome (HLHS).

Though congenital heart disease (CHD) occurs in only a small number of live-born infants (0.8%),[8,9] it is a leading cause of death in newborns. Hypoplastic left-heart syndrome (HLHS) was recently reported to affect 0.016% to 0.036% of all reported live-born infants, or 1.4% to 3.8% of CHD patients, with a 60%-70% male predominance. In siblings of these patients, HLHS occurs in 0.5% with a 2.2% risk of other forms of CHD.[10]

In a 1988 review of 83 autopsies of HLHS patients at the Children's Hospital of Philadelphia, genetic elements were found to be a determining factor of HLHS. Natwicz et al[11,12] reported that 4 cases had single gene defects, 9 had underlying chromosomal abnormalities, and 10 had one or more major extracardiac anomalies without an identifiable chromosomal disorder. In all, 28% had underlying chromosomal involvement—Turner's syndrome and Trisomies 21, 18, and 13. In 1990, Natwicz et al[13] reported congenital brain anomalies associated with HLHS. Of the 41 autopsies reviewed, 29% revealed some degree of central nervous system abnormality, 17% had specific anomalies (eg, agenesis of the corpus callosum), and 27% had microcephaly.

Infants suffering from HLHS may present with hypoplastic or absent left ventricle, aortic atresia, mitral atresia or stenosis, and hypoplastic or diminutive aorta.[8,14] A ventricular septal defect (VSD) may be associated with this syndrome. Recently HLHS has been categorized into four anatomic subtypes based on the morphology of the left heart valves: (1) aortic and mitral stenosis, (2) aortic and mitral atresia, (3) aortic atresia and mitral stenosis, and (4) aortic stenosis and mitral atresia.[1] Specific anatomy may also impede coronary perfusion, and, in the face of an obstructed interatrial connection, pulmonary vein dilatation may be present. In the case of HLHS, with its single functioning ventricle (right ventricle, RV, Fig. 1) systemic perfusion depends on an uninterrupted interatrial pathway through an atrial septal defect (ASD) and a right-to-left shunt through a patent ductus arteriosus (PDA). These anomalies are necessary to sustain life in that they provide systemic blood flow that would otherwise be essentially nonexistent.

The embryologic development[8] of the cardiovascular system is a complex process of biochemical events—a process that is yet to be fully understood. Before the end of the first trimester of gestation, the development of the heart is normally complete. However, a number of malformations can occur during this complex phase of embryonic metamorphosis. These malformations may be a direct or indirect result of many fac-

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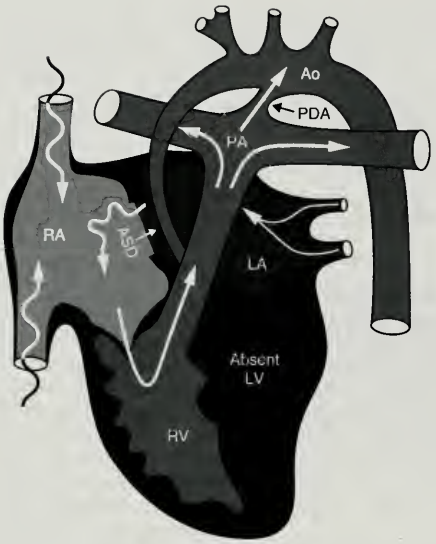

Fig. 1. This drawing shows the typical presentation of HLHS. Note the diminutive ascending aorta (Ao), atrial septal defect (ASD), absent left ventricle (LV), and patent ductus arteriosus (PDA). With a single functioning right ventricle (RV), systemic circulation is absolutely dependent upon a patent ductus arteriosus (PDA). RA, right atrium; LA, left atrium; PA, pulmonary artery.

tors including inflammatory responses to genetically mediated events, medications, and adverse environmental influences. Any untoward event that occurs during this period may cause abnormal morphogenesis of the fetal heart.

The patient with HLHS requires that adequate tissue perfusion be provided by PDA-dependent systemic blood flow ($\dot{Q}_s$). When the PDA closes, $\dot{Q}_s$ ceases, causing circulatory shock and metabolic acid production (due to reduced renal perfusion) to ensue. An increase in pulmonary blood flow ($\dot{Q}_p$) with a functionally absent left ventricle leads to elevated left-atrial pressure and subsequent pulmonary edema. The balance between excessive or decreased $\dot{Q}_p$ can be expressed by the ratio of pulmonary vascular resistance (PVR) to systemic vascular resistance (SVR). To prevent excessive low $\dot{Q}_p$ and the cascade of adverse events that may follow, palliative surgery is necessary, and a delicate balance of systemic and pulmonary blood pressures must be maintained.

Adequate $\dot{Q}_s$ is reasonably accurate evidence that the pulmonary vasculature is less 'flooded.' Measurements or estimations of cardiac output in these cases are inconclusive and have little bearing on whether $\dot{Q}_s$ is adequate. Cardiac output is the sum of pulmonary and systemic blood flow ($\dot{Q}_p + \dot{Q}_s$). Therefore, if $\dot{Q}_s$ increases and $\dot{Q}_p$ decreases by an equivalent amount, then systemic oxygen delivery declines without a change in cardiac output. However, a $\dot{Q}_p/\dot{Q}_s$ of 2 would indicate excessive pulmonary blood flow most likely secondary to decreased PVR. On the other hand, a $\dot{Q}_p/\dot{Q}_s$ of 0.5 could indicate excessive systemic blood flow due, most commonly, to an anatomic obstruction (eg, pulmonic stenosis) or an increase in PVR (Fig. 2). As PVR exceeds SVR, pulmonary blood flow is compromised, and, although adequate pulse pressures can be present, profound cyanosis ensues due to diminishing systemic oxygen delivery. Thus, it is imperative to monitor the strength or weakness of pedal pulses by palpation and oxyhemoglobin saturation by pulse oximetry (S_{pO_2}) to ascertain the adequacy of $\dot{Q}_s$ and oxygenation.

Without intervention, 95% of infants with HLHS die within the first month of life.[8,15] Two options are available for these patients—palliative reconstructive surgery or heart transplant.[12,16] The latter is not often a real option because of the emergency normally posed by these unstable infants. Patient size is an issue in organ procurement, rendering emergency transplantation impractical.[17] Thus, the only viable option for these patients is the surgical technique known as the Norwood Procedure.[14]

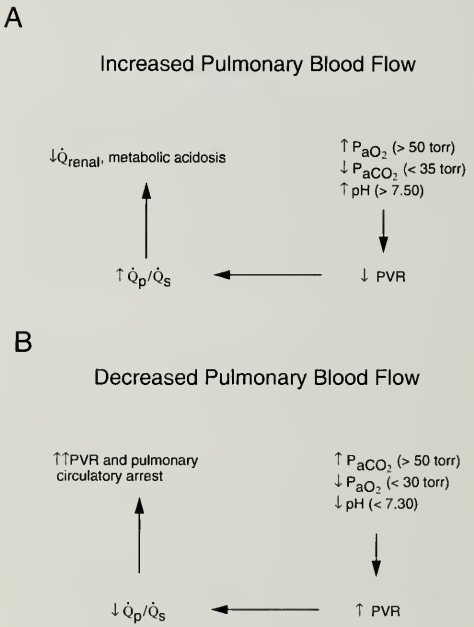

Fig. 2. Diagram showing the cascade of adverse cardiorespiratory events associated with increased (A) and decreased (B) pulmonary blood flow in patients who have undergone the Stage-1 Norwood Procedure. To avoid initiating the cascade, a delicate balance of blood-gas values must be maintained to ensure a proper balance of pulmonary and systemic blood flow. PVR, pulmonary vascular resistance; $\dot{Q}_p/\dot{Q}_s$, ratio of pulmonary-to-systemic perfusion.

Surgical Technique

The Norwood Procedure is performed in 3 stages. Stage I is a palliative procedure—ie, it is not a definitive correction of the defect. The second (hemi-Fontan) and third (completed Fontan) are designed to passively route venous blood flow to the pulmonary circulation, thus alleviating venous pressure from the right ventricle. These last two stages are typically done at 6 months and 18 months of age, respectively. As seen in Figure 3, adequate systemic and coronary perfusion are critically dependent upon the surgical procedure. In the first palliative stage[1,14,16] (Fig. 4), the pulmonary artery trunk is transected from its transverse branches and anastomosed, via a homograft patch, to the atretic aortic trunk. $\dot{Q}_S$ is thus maximized through a broader aortic access. In most cases a 3.0-5.0 mm Gore-Tex shunt is accurately placed to connect the descending aorta and the ipsilateral pulmonary artery, providing fixed pulmonary access. The PDA is ligated, and an unrestricted interatrial pathway is provided via atrial septectomy, to complete the first stage of the Norwood Procedure. With so many suture lines, a high risk of hemorrhage at the anastomosed sites exists. Extreme care should be taken in managing and handling these infants.

Case Presentation

A 3.5-kg Caucasian boy was delivered via cesarean section at 38-weeks gestation to a 27-year-old woman. Fetal

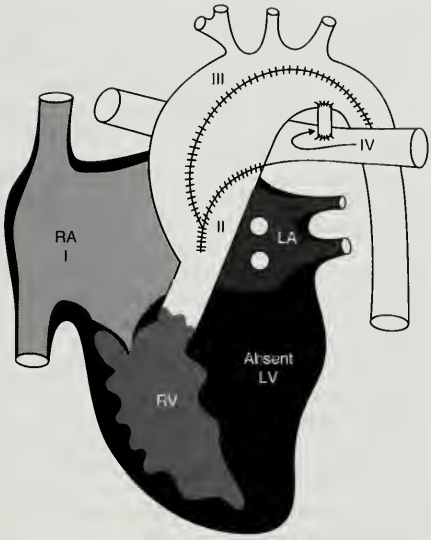

Fig. 3. This drawing illustrates the Stage-1 Norwood Procedure. With the pulmonary artery trunk (II) anastomosed to the ascending aorta (III), it is essential that PVR be under scrupulous control to avoid excessive pulmonary blood flow through the fixed pulmonary shunt (IV). RA, right atrium; RV, right ventricle; LV, left ventricle.

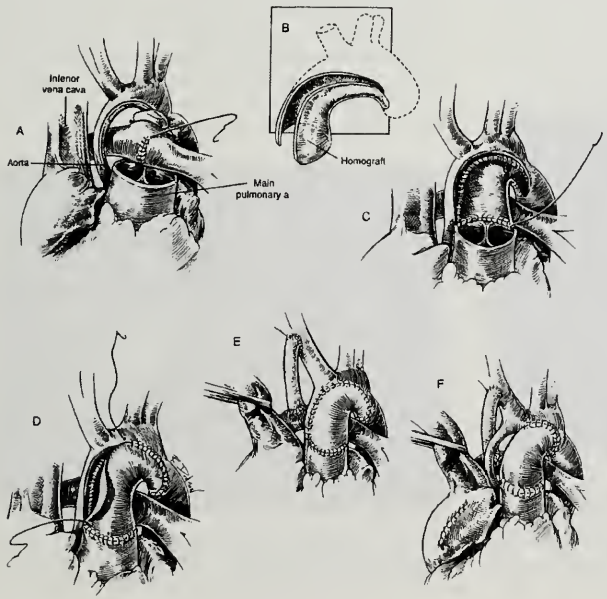

Fig. 4. Current techniques for first-stage palliation of the hypoplastic left heart syndrome. A, Incisions used for the procedure incorporate a cuff of aorta-wall allograft. The distal divided main artery may be closed by direct suture or with a patch. B, Magnification of the cuff of the aorta-wall allograft. C, The aorta-wall allograft is used to supplement the anastomosis between the proximal divided main pulmonary artery and the ascending aorta, aortic arch, and proximal descending aorta. D,E, The procedure is completed by an atrial septectomy and a 3.5-mm modified right Blalock shunt. F, When the ascending aorta is particularly small, an alternative procedure involves placement of a complete tube of arterial allograft. The tiny ascending aorta may be left in situ, as indicated, or implanted into the side of the reconstructed aorta (neoaorta). (Reprinted from Reference 1, with permission.)

echocardiogram had revealed the diagnosis of HLHS. The initial clinical examination revealed a severely cyanotic and poorly perfused ('shocky') infant in need of improved ventilation and systemic perfusion. Intubation was immediately accomplished following sedation and pharmacologic paralysis. Prostaglandin E_1 (PGE_1) was also administered to induce and maintain a PDA to ensure $\dot{Q}_S$.

PGE_1 has a variety of systemic effects.[18] The most important of these in this baby were relaxation of smooth muscle (particularly that of the ductus arteriosus), resultant vasodilatation, and inhibition of platelet aggregation. Due to the high rate at which PGE_1 is metabolized (80% elimination/cycle of pulmonary circulation), a continuous infusion must be administered to maintain the PDA and, thus, provide oxygenated blood for the systemic circulation.

The initial diagnosis of HLHS was confirmed by echocardiography (Fig. 5A), demonstrating right ventricular hypertrophy, a slight regurgitation via the tricuspid valve, a dilated pulmonary artery, a large PDA with adequate right-to-left shunt, a small atrial septal defect, the absence of a mitral valve and left ventricle, and a severely stenotic aorta. In Figure 5B the small lumen of the aorta can be seen and compared to the large pulmonary artery.

Expeditious transfer to a medical center capable of providing tertiary pediatric care is paramount to the perioperative prognosis of these infants.[19] During transport and following admission to our center, the patient was ventilated with an F_{DO_2} 0.21, mechanical respiratory rate 40, peak inspiratory pressure (PIP) 24 cm H_2O, and positive end-expiratory pressure (PEEP) 4 cm H_2O. Arterial blood-gas values were pH 7.43, P_{aCO_2} 30 torr, and a P_{aO_2} 27 torr. S_{pO_2} was maintained in the 70-75% range, which was adequate for this infant. The goal was to maintain the lowest S_{pO_2} that would support adequate $\dot{Q}_p$—as evidenced by the strength of the peripheral pulses.

Fig. 5A. A parasternal long-axis echocardiograph of HLHS. Notice the classic left-ventricular hypoplasty (LV) and the hypertrophic right ventricle (RV). The atrial septal defect between the right (RA) and left (LA) atria is the sole provider of oxygenated pulmonary blood return. The patient is entirely dependent on his RV, patent ductus arteriosus, and atrial septal defect (ASD) for cardiac output.

Fig. 5B. A parasternal short axis view demonstrating the incapacity of the native aorta to provide adequate blood flow even in the face of an existing small left ventricle.

Postoperatively, the infant demonstrated adequate systemic perfusion with normal ventilation. As seen in Figure 5C a broader outflow tract is provided by the reconstructed aorta (neoaorta). However, within the hour, PVR began to decline, effecting a deterioration in $\dot{Q}$, and systemic oxygen delivery (indicated by diminished peripheral perfusion, reduced pulse pressures, and, most critically, a drop in urine output). Metabolic acid production ensued secondary to decreased renal perfusion. To more efficiently control PVR, the patient was prepared for hypoxic-gas therapy. Reduced inspired oxygen delivery is achieved by the addition of nitrogen (N_2) to the inspired gas, reducing the F_{DO_2} to less than that of room air. As the patient's perfusion became progressively worse, systemic blood

Fig. 5C. Postoperative Stage-1 Norwood Procedure echocardiograph showing the newly formed neoaorta (PA, pulmonary artery) and its outflow track supplied by the right ventricle (RV).

pressure began to drop, S_{pO_2} decreased to < 50%, and central venous pressure (CVP), an indirect indicator of cardiac output, also fell. The general picture revealed progressive systemic blood-flow reduction and flooding of the pulmonary circulatory bed. The ratio of PVR to SVR was low. The patient was immediately given an F_{DO_2} of 0.19, and, although PIP was only as high as necessary to prevent atelectasis, P_{aCO_2} remained undesirably low (35 torr). To avoid administering a more hypoxic gas, CO_2 was administered (P_{DCO_2} 15 torr) to facilitate a further increase in PVR. With the reduction of F_{DO_2} and the increase in P_{DCO_2}, the patient's $\dot{Q}_s$, S_{pO_2}, and CVP all returned to acceptable levels. By administering $F_{DO_2} < 0.21$, the pulmonary vascular bed reacted in a normal physiologic manner, increasing PVR. The resulting PVR/SVR was more compatible with parallel circulation principles. However, in time it was evident by the repeated decline in peripheral perfusion that further circulatory manipulation was needed. Hypercarbic-gas therapy was successful in increasing PVR, eliminating the need for further potentially risky reductions in F_{DO_2}.

Following extubation on the postoperative Day 4, the child seemed comfortable His peripheral pulses were acceptably strong, and S_{pO_2} was in the 60-70% range. However, the child soon began to display signs of systemic instability marked by a gradual decline in SBP, pulse pressures, and urine output. In an effort to restore an adequate pulmonary-systemic blood-flow ratio, the patient was again treated with F_{DO_2} 0.18 and P_{DCO_2} 15 torr via oxygen hood. This tactic initially resulted in elevated PVR and enhanced pulse pressures. However, intubation and mechanical ventilation were re-instituted due to an increase in observed work of breathing, and failure to maintain adequate lung volume. On postoperative Day 28, this patient was extubated and, shortly thereafter, discharged home on room air.

Discussion

Neonatal respiratory therapists are accustomed to caring for newborns with PPHN. This unbalanced pulmonary-to-systemic circulation is compatible with fetal life, but fatal to the newborn. In the ineffectual lungs of the human fetus, high PVR and low SVR work well for an organism that depends on its host for tissue oxygenation and respiration. However, after the infant is delivered, these resistances must reverse to meet the demands of extrauterine life. With the anomalous circulatory dynamics present in infants who have undergone the Norwood Procedure, a low PVR:SVR is fatal. The accepted practice of treating PPHN—promoting pulmonary vasodilatation to maximize pulmonary blood flow—can be devastating for these patients.

Following the Stage-1 Norwood Procedure there is but one pathway to the pulmonary circulation: the aorta-pulmonary artery shunt. The flow through the shunt is governed by several factors: (1) shunt length, (2) shunt diameter, (3) resistance at both ends, and (4) fluid viscosity. Because shunt length and diameter remain constant, these factors can be omitted from the equation. The remaining two factors, blood viscosity and blood pressures, can be manipulated. Allowing low viscosity circulation increases $\dot{Q}_p$ but is a more tedious system to control. A more viscous circulation promotes better blood-flow control and increases oxygen carrying capacity. So as not to overload the right ventricle, PVR:SVR is maintained slightly higher than normal, but not so high as to impede $\dot{Q}_p$. We expect blood flow to follow the path of least resistance and thus improve systemic perfusion. However, this is rarely the case.

As implied in the Introduction, PVR is normally considerably lower than SVR in extrauterine life, and this relationship persists in most patients after the Norwood Procedure. Recalling the effect of P_{aO_2} and P_{aCO_2} on PVR (Fig. 2), it is reasonable to expect that a reduced P_{aO_2}, and an increased P_{aCO_2} would promote pulmonary vasoconstriction and enhance systemic perfusion. However, we do not entirely understand how or why PVR reacts to these gases as it does. It has been suggested that the increased P_{aCO_2}–not decreased pH level–is responsible for the pulmonary vasoconstriction, even though low pH values have been strongly linked with increased PVR. Because acid-base balance is largely stable in a mechanically ventilated infant, it is unlikely that pH is the effector of pulmonary vasoconstriction. Furthermore, it has been noted that there is a substantial increase in plasma epinephrine and norepinephrine levels during periods of elevated P_{aCO_2}, suggesting that the effect is an indirect one caused by an adjustment of PVR:SVR due to increased SVR.[7]

Conclusion

Throughout the 28-day period of this case, numerous gas manipulations were required and successfully performed to maintain systemic perfusion in this patient. Hypoxic-gas therapy was administered within an F_{DO_2} range of 0.12-0.20 (mean 0.18), and P_{DCO_2} range of 1-20 torr (mean 9 torr). This regimen produced optimal levels of P_{aO_2} (30-35 torr) and P_{aCO_2} (45-50 torr).

Our experience may be useful in developing guidelines that are effective in increasing PVR in patients with parallel circulation. Of all the interventions (both pharmacologic and ventilatory) undertaken in the management of this infant after the Norwood Procedure, the most successful for restoring the pulmonary-systemic blood-flow balance were hypoxic- and hypercarbic-gas therapy. The 28-day postoperative course of the infant whose case we report has been a valuable experience. Since then, we have managed several infants with HLHS using a similar protocol, and these have experienced a similar outcome. This case demonstrates that it is possible to successfully maintain adequate alveolar ventilation and lung volume and, at the same time, stabilize PVR:SVR using hypoxic- and hypercarbic-gas therapy in an infant with HLHS who has undergone the Stage-1 Norwood Procedure.

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AARC Clinical Practice Guideline

Defibrillation during Resuscitation

DDR 1.0 PROCEDURE:

Therapeutic use of electrical current for defibrillation.

DDR 2.0 DESCRIPTION:

Electrical therapy for the purposes of this guideline encompasses all care necessary for defibrillation during cardiac arrest on all patients with ventricular fibrillation or pulseless ventricular tachycardia. This includes the use of conventional defibrillators and automated (automatic or semi-automatic) external defibrillators (AEDs).

DDR 3.0 SETTING:

This guideline applies to a variety of settings including but not limited to hospitals, long-term facilities, outpatient clinics, rehabilitation centers, skilled nursing facilities, and pre- and interhospital transport.

DDR 4.0 INDICATIONS:

4.1 Cardiac arrest due to or resulting in ventricular fibrillation.[1-3]
4.2 Pulseless ventricular tachycardia.

DDR 5.0 CONTRAINDICATIONS:

Defibrillation is contraindicated when:

5.1 the patient's desire not to be resuscitated has been clearly expressed and documented in the patient's medical record or other legal document;[4-6]
5.2 continued resuscitation is determined to be futile by the treating physician;[7-13]
5.3 immediate danger to the rescuers is present due to the environment, patient's location, or patient's condition.

DDR 6.0 PRECAUTIONS/HAZARDS:

6.1 AEDs may be hazardous in patients weighing 90 lb or less.[14]
6.2 Superficial arcing of the current along the chest wall can occur as a consequence of the presence of conductive paste or gel between the paddles.[15]
6.3 Malfunction of permanent pacemakers can result from placing defibrillator pads or paddles near the pacemaker.[16,17]
6.4 Defibrillation in the absence of an ECG rhythm (ie, 'blind defibrillation') is rarely necessary today because of the almost universal availability of AEDs equipped with monitoring capabilities and diagnostic algorithms. In rare circumstances when electrocardiographic (ECG) monitoring cannot be implemented in a timely fashion, the experienced practitioner may elect to apply blind defibrillation to a pulseless, comatose patient.[14-15]
6.5 The aluminized backing on some transdermal systems can cause electric arcing during defibrillation, with explosive noises, smoke, visible arcing, patient burns, and impaired transmission of current;[18-20] therefore, patches should be removed before defibrillation.
6.6 A shock can be accidentally delivered to other rescuers.[21]
6.7 Pulse checking between sequential shocks of AEDs delays rapid identification of persistent ventricular fibrillation, interferes with assessment capabilities of the devices, and increases the possibility of operator error.[14]
6.8 The initial three shocks should be delivered in sequence, without interruption for CPR, medication administration, or pulse checks.[22,23]
6.9 Delays in delivering shocks for ventricular fibrillation and pulseless ventricular tachycardia after defibrillator arrival should be avoided.[1,2,24]

6.10 If transthoracic impedance is high, a low energy shock (< 100 J) may fail to generate enough current to achieve successful defibrillation.[25-27]
6.11 Alcohol should never be used as coonducting material for paddles because serious burns can result.[28]
6.12 Attention must be paid to factors influencing total and transthoracic impedance.[14,25, 27,28]
6.12.1 Paddle electrode pressure
6.12.2 The use of an appropriate conductive medium that can withstand high current flow.[29]
6.12.3 The use of hand-held paddles greater than 8 cm in diameter for adults or children weighing more than 40 kg.[30]
6.12.4 Electrode placement
6.12.5 Time interval between shocks
6.12.6 Distance between electrodes (size of the chest)
6.12.7 Electrode/paddle size[30]
6.12.8 Energy selected
6.12.9 Paddle-skin electrode material
6.12.10 Number of previous shocks
6.12.11 Phase of ventilation
6.12.12 Diaphoretic patients should be dried to prevent contact problems with adhesive defibrillation pads and/or electrodes.[14]
6.13 AEDs may be hazardous in an oxygen-enriched environment.[31]

DDR 7.0 LIMITATIONS OF PROCEDURE:

Despite appropriate and technically adequate use of electrical therapy, outcome may not be successful because of patient's underlying condition and deterioration of the patient's cardiac status.

7.1 Response is poor in subjects with extremely low core temperatures, and shocks should be limited to three until temperature has risen above 86 °F.[30] Warming may improve success.[32]
7.2 Subjects whose cardiac arrest occurs as a direct result of trauma may not respond to defibrillation.[32]
7.3 The patient must not move or be moved while analysis is occurring when the automated or semi-automated defibrillator is used. (Compressions must be stopped, and if the patient is in a vehicle, it must not be moving).

DDR 8.0 ASSESSMENT OF NEED:

8.1 Before arrival of defibrillator: The patient should be assessed for lack of responsiveness, apnea, and pulselessness, and help should be summoned if needed.[14,33,34]
8.2. After arrival of defibrillator: The patient should be evaluated immediately for the presence of ventricular fibrillation or ventricular tachycardia by the operator (conventional) or the defibrillator (automated or semi-automated). Inappropriate defibrillation can cause harm.[33,35]

DDR 9.0 ASSESSMENT OF PROCESS & OUTCOME:

9.1 Equipment management issues. Use of standard checklists can improve defibrillator dependability.[36]
9.2 Defibrillation process issues
9.2.1 System access[34]
9.2.2 Response time[37]
9.2.3 First-responder actions[37,38]
9.2.4 Adherence to established algorithms[39]
9.2.5 Patient selection and outcome
9.2.6 First responder authorization to defibrillate[14,40]

DDR 10.0 RESOURCES:

10.1 Personnel: A high percentage of patients in nontraumatic cardiac arrest are in ventricular fibrillation within the first few minutes after their collapse. As time after arrest increases the likelihood of a successful outcome decreases rapidly.[41,42] Within the hospital, all personnel who have direct patient contact should be trained in CPR and early defibrillation as first responders.[43] Early defibrillation as a standard has been expanded to include the use of AEDs by first responders trained in basic life support (BLS), for both prehospital and in-hospital cardiac arrest due to ventricular fibrillation.
10.1.1 Level I:
10.1.1.1 Training—Automated external defibrillation is a basic life support (BLS) skill and should be incorporated into BLS training programs for all hospital personnel who are expected to re-

spond to a cardiac arrest and who have access to AEDs. Because AEDs are sensitive and specific in recognizing shockable rhythms,[44] there is no need to train the operator to recognize ventricular fibrillation or ventricular tachycardia or any dysrhythmia. Less training time is needed to learn how to use an AED than to learn dysrhythmia recognition.[43] There should be continuing education and documentation of competency at least every 90 days in the employee's departmental folder.[33,35,45]

10.1.1.2 Responsibilities—Designated first responders must be able to recognize that the patient is unresponsive, apneic, and pulseless. They should be able to attach automated defibrillator electrodes, operate AEDs, and complete an AED checklist at least every shift.[46]

10.1.1.3 Level I: All health-care providers who have direct patient care responsibilities and may be the first responder to patients in cardiac arrest are considered Level I caregivers. No special professional credential is necessary to qualify as Level I, by this definition.

10.1.2 Level II:

10.1.2.1 Training—Level II personnel should be trained, evaluated, and retrained as necessary in use of automated and conventional defibrillators, emergency cardiac care, and advanced cardiac life support (ACLS) at frequent intervals. The time between defibrillation practice sessions should be limited to 90 days or less.[14,44,47]

10.1.2.2 Responsibilities—Level II personnel have the skills of Level I personnel plus the following capabilities: (1) advanced ECG monitoring and dysrhythmia recognition, (2) capability to deliver shocks with automated and conventional external defibrillators.

10.1.2.3 Credentials—A Level II health professional should be an RRT, RN, MD, or DO, or hold an equivalent credential and should hold a current AHA ACLS or pediatric advanced life support (PALS) course-completion card or have evidence of having completed a similar recognized training program.

10.2 Equipment:

10.2.1 Automated or semi-automated external defibrillators must be able to determine whether defibrillation is appropriate and advise either delivery of or deliver appropriate shocks. The defibrillators should be maintained in accordance with manufacturer's specifications.[33,35,45]

10.2.2 Conventional defibrillators should be maintained in accordance with manufacturer's specifications. Batteries should be maintained in a state of full charge and reconditioned appropriate.[32,45]

10.2.3 Defibrillator checklists should be used to reduce defibrillator malfunctions.[15] Users should be competent in the proper use of checklists to reduce defibrillator malfunctions.[15,36]

DDR 11.0 MONITORING:

11.1 Resuscitation process—Properly performed defibrillation has been shown to improve patient outcome. The most important determinant of survival in adult out-of-hospital ventricular fibrillation is defibrillation. Continuous monitoring of the process identifies components needing improvement. Among these components are response time, witnessed versus unwitnessed arrest, CPR performance, time-to-first defibrillation attempt, return of spontaneous circulation, complication rate, equipment function, equipment maintenance, and equipment availability.[1,2,36,48,49]

11.2 Equipment—All maintenance should be documented and records preserved. Included in documentation should be routine checks of energy output, condition of batteries, proper functioning of monitor and recorder, and presence of disposables needed for function of defibrillator, including electrodes and defibrillation pads. Defibrillators should be checked each shift[46] for presence, condition and function of cables and paddles; presence of defib-

rillating and monitoring electrodes, paper, and spare batteries (as applicable); and charging, message/light indicators, monitors, and ECG recorder (as applicable).[33,35]

11.3 Training—Records should be kept of initial training and continuing education of all personnel who perform defibrillation as part of their professional activities.

DDR 12.0 FREQUENCY/AVAILABILITY/ DURATION:

Personnel who respond to cardiac arrests should be trained to operate, equipped with, and permitted to operate a defibrillator.[14] No other therapeutic intervention, including setting up oxygen delivery systems, suction equipment, advanced airway procedures, intravenous lines, or mechanical CPR devices, should take precedence over or be routinely performed when a defibrillator is available and defibrillation is indicated.[14]

DDR 13.0 INFECTION CONTROL:

13.1 Implement Universal Precautions and tuberculosis control measures.[49,50]

13.2 Observe all infection control guidelines posted for the patient.

13.3 Disinfect all equipment to be reused on other patients.

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AARC Clinical Practice Guideline

Management of Airway Emergencies

MAE 1.0 PROCEDURE:

Recognition of signs of an impending or actual airway emergency. Initial treatment and continued management of airway emergencies to minimize the likelihood of adverse outcomes, in adults, children, and infants.

MAE 2.0 DESCRIPTION/DEFINITION:

Management of airway emergencies (MAE) for the purpose of this guideline encompasses all care necessary to deal with sudden and often life-threatening events affecting natural and artificial airways and involves the identification, assessment, and treatment of patients in danger of losing or not being able to maintain an adequate airway, including the newborn. This includes (1) identification of the causes of airway emergencies; (2) management of airway emergencies prior to tracheal intubation; (3) use of adjunctive equipment and special techniques for establishing, maintaining, and monitoring effective ventilation; (4) translaryngeal tracheal intubation, including nasal and oral tracheal intubation; (5) transtracheal catheter ventilation, (6) percutaneous dilational cricothyrotomy, and; (7) surgical cricothyrotomy.

MAE 3.0 SETTING:

The guideline applies to a variety of settings including but not limited to hospitals and pre- or interhospital transport.

MAE 4.0 INDICATIONS:

4.1 Conditions requiring management of the airway, in general, are impending or actual (1) airway compromise, (2) respiratory failure, and (3) need to protect the airway. Specific conditions include but are not limited to

- **4.1.1** Airway emergency prior to endotracheal intubation
- **4.1.2** Obstruction of the artificial airway
- **4.1.3** Apnea
- **4.1.4** Acute traumatic coma[1]
- **4.1.5** Penetrating neck trauma[2]
- **4.1.6** Cardiopulmonary arrest and unstable dysrhythmias[3]
- **4.1.7** Severe bronchospasm[4-8]
- **4.1.8** Severe allergic reactions with cardiopulmonary compromise[9,10]
- **4.1.9** Pulmonary edema[11,12]
- **4.1.10** Sedative or narcotic drug effect[13]
- **4.1.11** Foreign body airway obstruction[3]
- **4.1.12** Choanal atresia in neonates[14]
- **4.1.13** Aspiration
- **4.1.14** Risk of aspiration
- **4.1.15** Severe laryngospasm[15]
- **4.1.16** Self-extubation[16,17]

4.2 Conditions requiring emergency tracheal intubation include, but are not limited to

- **4.2.1** Persistent apnea
- **4.2.2** Traumatic upper airway obstruction (partial or complete)[18-20]
- **4.2.3** Accidental extubation of the patient unable to maintain adequate spontaneous ventilation[16,17]
- **4.2.4** Obstructive angioedema (edema involving the deeper layers of the skin, subcutaneous tissue, and mucosa)[21-23]
- **4.2.5** Massive uncontrolled upper airway bleeding[2,24]
- **4.2.6** Coma with potential for increased intracranial pressure[25]
- **4.2.7** Infection-related upper airway obstruction (partial or complete)
  - **4.2.7.1** Epiglottitis in children or adults[26,27]
  - **4.2.7.2** Acute uvular edema[28]
  - **4.2.7.3** Tonsillopharyngitis or retropha-

ryngeal abscess[29]

4.2.7.4 Suppurative parotitis[30]

4.2.8 Laryngeal and upper airway edema[31]

4.2.9 Neonatal- or pediatric-specific

4.2.9.1 Perinatal asphyxia[32,33]

4.2.9.2 Severe adenotonsillar hypertrophy[34,35]

4.2.9.3 Severe laryngomalacia[36,37]

4.2.9.4 Bacterial tracheitis[38-40]

4.2.9.5 Neonatal epignathus[41,42]

4.2.9.6 Obstruction from abnormal laryngeal closure due to arytenoid masses[43]

4.2.9.7 Mediastinal tumors[44]

4.2.9.8 Congenital diaphragmatic hernia[45]

4.2.9.9 Presence of thick and/or particulate meconium in amniotic fluid[46-48]

4.2.10 Absence of airway protective reflexes

4.2.11 Cardiopulmonary arrest

4.2.12 Massive hemoptysis[49]

4.3 The patient in whom airway control is not possible by other methods may require surgical placement of an airway (needle or surgical cricothyrotomy).[20,50,51]

4.4. Conditions in which endotracheal intubation may not be possible and in which alternative techniques may be used include but are not limited to

4.4.1 restriction of endotracheal intubation by policy or statute;

4.4.2 difficult or failed intubation in the presence of risk factors associated with difficult tracheal intubations[52] such as

4.4.2.1 Short neck,[53] or bull neck[54]

4.4.2.2 Protruding maxillary incisors[53]

4.4.2.3 Receding mandible[53]

4.4.2.4 Reduced mobility of the atlanto-occipital joint[55]

4.4.2.5 Temporomandibular ankylosis[55]

4.4.2.6 Congenital oropharyngeal wall stenosis[56]

4.4.2.7 Anterior osteophytes of the cervical vertebrae, associated with diffuse idiopathic skeletal hyperostosis[57]

4.4.2.8 Large substernal and/or cancerous goiters[58]

4.4.2.9 Treacher-Collins syndrome[59]

4.4.2.10 Morquio-Brailsford syndrome[60]

4.4.2.11 Endolaryngeal tumors[61]

4.4.3 when endotracheal intubation is not immediately possible

MAE 5.0 CONTRAINDICATIONS:

Aggressive airway management (intubation or establishment of a surgical airway) may be contraindicated when the patient's desire not to be resuscitated has been clearly expressed and documented in the patient's medical record or other valid legal document.[62-64]

MAE 6.0 PRECAUTIONS/HAZARDS AND/OR COMPLICATIONS:

The following represent possible hazards or complications related to the major facets of management of airway emergencies:

6.1 Translaryngeal intubation or cricothyrotomy is usually the route of choice. It may be necessary occasionally to use a surgical airway. Controversy exists as to whether intubation is hazardous in the presence of an unstable injury to the cervical spine. In one series the incidence of serious cervical spine injury in a severely injured population of blunt trauma patients was relatively low, and commonly used methods of precautionary airway management rarely led to neurologic deterioration.[65-67]

6.1.1 Failure to establish a patent airway[68-70]

6.1.2 Failure to intubate the trachea[68,69]

6.1.3 Failure to recognize intubation of esophagus[25,68,71-81]

6.1.4 Upper airway trauma, laryngeal, and esophageal damage[82]

6.1.5 Aspiration[70,74,82,83]

6.1.6 Cervical spine trauma[67,84,85]

6.1.7 Unrecognized bronchial intubation[25,68,72,82,86,87]

6.1.8 Eye injury[70]

6.1.9 Vocal cord paralysis[88]

6.1.10 Problems with ETT tubes

6.1.10.1 Cuff perforation[89]

6.1.10.2 Cuff herniation[89]

6.1.10.3 Pilot-tube-valve incompetence[90]

6.1.10.4 Tube kinking during biting[70,89]

6.1.10.5 Inadvertent extubation[17,25,68,72,86,91-93]

6.1.10.6 Tube occlusion[17,72,82,89,93,94]

6.1.11 Bronchospasm[68,70,74]

6.1.12 Laryngospasm[72]

6.1.13 Dental accidents[70]

6.1.14 Dysrhythmias[94]

6.1.15 Hypotension and bradycardia due to vagal stimulation[94]

6.1.16 Hypertension and tachycardia[94,95]

6.1.17 Inappropriate tube size[89,96-99]

6.1.18 Bleeding

6.1.19 Mouth ulceration[82]

6.1.20 Nasal-intubation specific

6.1.20.1 Nasal damage including epistaxis

6.1.20.2 Tube kinking in pharynx

6.1.20.3 Sinusitis[100-102] and otitis media

6.1.21 Tongue ulceration

6.1.22 Tracheal damage including tracheoesophageal fistula, tracheal innominate fistula, tracheal stenosis, and tracheomalacia[103-107]

6.1.23 Pneumonia[108]

6.1.24 Laryngeal damage with consequent laryngeal stenosis,[82,101,107,109,110] laryngeal ulcer, granuloma, polyps, synechia

6.1.25 Surgical cricothyrotomy or tracheostomy specific[111,112]

6.1.25.1 Stomal stenosis[82,113]

6.1.25.2 Innominate erosion[113]

6.1.26 Needle cricothyrotomy specific[114-118]

6.1.26.1 Bleeding at insertion site with hematoma formation

6.1.26.2 Subcutaneous and mediastinal emphysema[117]

6.1.26.3 Esophageal perforation

6.2 Emergency ventilation

6.2.1 Inadequate oxygen delivery[119-121]

6.2.2 Hypo- or hyperventilation[122-124]

6.2.3 Gastric insufflation and/or rupture[125,126]

6.2.4 Barotrauma[127-129]

6.2.5 Hypotension due to reduced venous return secondary to high mean intrathoracic pressure[130-132]

6.2.6 Vomiting and aspiration[125]

6.2.7 Prolonged interruption of ventilation for intubation[3,126]

6.2.8 Failure to establish adequate functional residual capacity in the newborn[133-135]

6.2.9 Movement of unstable cervical spine (more than by any commonly used method of endotracheal intubation).[136]

6.2.10 Failure to exhale due to upper airway obstruction during percutaneous transtracheal ventilation.[118,136]

MAE 7.0 LIMITATIONS OF PROCEDURE:

Despite adequate management of airway emergencies, desired outcome may not be achieved because of the patient's underlying condition and progression of the process leading to the need for emergency airway management.

MAE 8.0 ASSESSMENT OF NEED:

The need for management of airway emergencies is dictated by the patient's clinical condition. Careful observation, the implementation of basic airway management techniques, and laboratory and clinical data should help determine the need for more aggressive measures.

Specific conditions requiring intervention include

8.1 Inability to adequately protect airway (eg, coma, lack of gag reflex, inability to cough) with or without other signs of respiratory distress.

8.2 Partially obstructed airway. Signs of a partially obstructed upper airway include ineffective patient efforts to ventilate, paradoxical respiration, stridor, use of accessory muscles, patient's pointing to neck, choking motions, cyanosis, and distress. Signs of lower airway obstruction may include the above and wheezing.

8.3 Complete airway obstruction. Respiratory efforts with no breath sounds or suggestion of air movement are indicative of complete obstruction.

8.4 Apnea. No respiratory efforts are seen. May be associated with cardiac arrest.

8.5 Hypoxemia, hypercarbia, and/or acidemia seen on arterial blood gas analysis, oximetry or exhaled gas analysis.

8.6 Respiratory distress. Elevated respiratory rate, high or low ventilatory volumes, and signs

of sympathetic nervous system hyperactivity may be associated with respiratory distress.

MAE 9.0 ASSESSMENT OF PROCESS AND OUTCOME:

Timely intervention to maintain the patient's airway can improve outcome in terms of survival and level of function. Under rare circumstances, maintenance of an airway by nonsurgical means may not be possible. Despite optimal maintenance of the airway, patient outcomes are affected by patient-specific factors. Lack of availability of appropriate equipment and personnel may adversely affect patient outcome. Monitoring and recording are important to the improvement of the process of emergency airway management. Some aspects (eg, frequency of complications of tracheal intubation or time to establishment of a definitive airway) are easy to quantitate and can lead to improvement in hospitalwide systems. Patient condition following the emergency should be evaluated from this perspective.

MAE 10.0 RESOURCES:

10.1 Personnel: All health professionals should be trained, evaluated at frequent intervals, and retrained as necessary in the skills of emergency clearance of foreign body airway obstruction and airway secretions. Health professionals who are primary members of resuscitation teams in acute care hospitals should be skilled in advanced management of airway emergencies, emergency cardiac care (ECC), and advanced cardiac life support (ACLS).[137]

Emergency response system—a designated resuscitation team should be continuously available (24 hours/day, 7 days/week) to assist with the management of airway emergencies. Team members should be notified simultaneously. All hospital workers must know how to activate the hospital's emergency response system.[137]

10.1.1 Level I

10.1.1.1 Training—all Level I personnel should be trained, evaluated by performance, and retrained as necessary in clearance of foreign-body airway obstruction, emergency airway-secretion evacuation techniques, and basic life support (BLS) at frequent intervals that do not exceed one year. Retraining should focus on identified deficiencies.

10.1.1.2 Responsibilities—Level I personnel are health professionals who assist the primary (Level II) members of the health-care team. They should be capable of assisting Level II personnel by (1) assessing patients for airway emergencies, respiratory, and/or cardiac arrest, (2) activating the resuscitation team, (3) administering BLS, (4) clearing the airway of foreign-body obstruction or material with the potential for obstruction, (5) providing mouth-to-mask ventilation, (6) assisting with tracheal intubation, (7) attaching pulse oximeter and capnograph, (8) moving adjunct airway equipment to the scene, (9) collecting arterial blood for analysis, (10) making a written record of resuscitation efforts.

10.1.1.3 Credentials—Level I health professionals should hold one or more of the following or equivalent credentials: RRT, CRTT, RN, MD, or DO; and have current BLS health-care provider-course completion card from the American Heart Association or a similar equivalent organization. Health professionals and hospital personnel not holding one of these Level I credentials should at a minimum be capable of assessing the patient for foreign-body airway obstruction, activating the resuscitation team, and administering BLS until the team arrives.

10.1.2 Level II

10.1.2.1 Training—Level II personnel should be trained, evaluated by performance, and retrained as necessary in advanced management of airway emergencies—ACLS and/or pediatric advanced life support (PALS) and/or neonatal resuscitation program (NRP)—as appropriate at intervals that should not exceed 1 year. To maintain operator competence, certain procedures (eg, endotracheal intubation) need to be reinforced

as often as every 3 months. Retraining should focus on identified deficiencies.

10.1.2.2 Responsibilities—Level II health professionals should be capable of serving as primary members of the resuscitation team. They may respond not only to airway emergency calls in their work areas but also to other areas of the hospital. They are skilled in the use of all adjunctive equipment and special techniques for ECC/ACLS, eg, establishing, maintaining, and monitoring effective ventilation and circulation as described in more detail in the AARC Clinical Practice Guideline: Resuscitation in the Acute Care Hospital.[137] They have the skills of Level I personnel and also the following capabilities: (1) advanced ECG monitoring and dysrhythmia recognition, (2) tracheal intubation and airway stabilization; (3) establishing ventilation via transtracheal catheter and cricothyrotomy; (4) emergency treatment of tension pneumothorax or hemothorax with large bore needle; (5) preparing patients for emergency transport; (6) use of continuous and transport mechanical ventilators, (7) evaluating oxygenation, ventilation and acid-base balance from blood gas reports.[138-141]

10.1.2.3 Credentials—Level II health professionals should hold one or more of the following credentials: RRT, RN, MD, or DO; and current ACLS, PALS, and/or NRP course-completion card from the American Heart Association or a similar equivalent association.

10.2 Equipment should be rapidly available and functional. Durability, portability, reliability, and cost should be considered

10.2.1 Ventilation devices should comply with the recommendations made in the AARC Guideline: Resuscitation in the Acute Care Hospital.[137]

10.2.2 Airway management devices should comply with the recommendations made in the AARC Guideline: Resuscitation in the Acute Care Hospital.[137] Some other airway devices are available (eg, laryngeal mask airway, esophageal obturator airway, combination esophageal-tracheal tubes) and may be acceptable and useful although they do not provide the airway control and protection afforded by an endotracheal tube.[142]

10.2.2.1 The laryngeal mask airway (LMA) provides a low-pressure seal around the glottis. Although its size effectively prohibits its being inserted into either the trachea or the esophagus, it does not reliably protect the airway from aspirated gastric contents. It may cause less airway trauma than the endotracheal tube and less cardiovascular instability. Available in sizes 1-4, it works in children as well as adults. It may be easier to insert in patients with higher Mallampati classifications.[69,143-151]

10.2.2.2 The esophageal obturator airway/esophageal gastric tube airway (EOA/EGTA) are considered together. Although researchers originally claimed it required less time for training than the endotracheal tube, this may not be the case. It is still widely used, primarily in prehospital care of adults. There have been several reports of the effectiveness of the EOA, but comparison is difficult due to variability in patients and in the medical supervision of the systems. Complications have been reported in many studies. While some believe it to be a useful second-line airway adjunct (for keeping gas out of the stomach and stomach contents out of the pharynx), others feel that the time used for training in EOA insertion would be better spent in training for endotracheal intubation or, failing that, placing more emphasis on basic airway maintenance and ventilation.[152-163]

10.2.2.3 The pharyngeotracheal lumen airway (PTL) is a double-lumen tube that is inserted blindly into the pharynx. After the position of the tube has been assessed, the patient is ventilated through the appropriate lumen. A large pharyngeal balloon seals the airway and a smaller secondary balloon is then inflated. The published complication

rate is low, but there has been relatively little evaluation of this device.[164,165]

10.2.2.4 The esophageal tracheal Combitube (ETC) is the newest airway device to be developed. It is similar to the PTL in that it is a double-lumen tube that is inserted blindly into the oropharynx; the position of the tube is assessed; and the patient is ventilated through the appropriate lumen. It has both a low reported rate of complications and few published evaluations.[24,166-172]

10.2.2.5 A 12-16 gauge intravenous catheter-over-the-needle device is used to initiate transtracheal catheter ventilation with the breathing mixture supplied at high pressure (30-50 psi). Because exhalation with this device must occur passively through the upper airway, CO_2 excretion is usually inadequate. This technique has potential for providing oxygen to the patient with a partially obstructed airway.

10.2.2.6 Percutaneous dilational cricothyrotomy is performed by making a small vertical incision and advancing a cricothyrotomy tube over a guidewire and dilator.[173] If the catheter is improperly placed or becomes dislodged, ventilation and oxygenation will fail and barotrauma will occur (eg, massive subcutaneous emphysema, pneumomediastinum, pneumothorax, bleeding).

10.2.2.7 Surgical cricothyrotomy requires experience, skill, and specialized equipment and may be facilitated by a tracheal dilator or tracheal hook and a standard tracheostomy or endotracheal tube.

MAE 11.0 MONITORING:

11.1 Patient

11.1.1 Clinical signs—continuous observation of the patient and repeated clinical assessment by a trained observer provide optimal monitoring of the airway. Special consideration should be given to the following:[174]

11.1.1.1 Level of consciousness

11.1.1.2 Presence and character of breath sounds

11.1.1.3 Ease of ventilation

11.1.1.4 Symmetry and amount of chest movement

11.1.1.5 Skin color and character (temperature and presence or absence of diaphoresis)

11.1.1.6 Presence of upper airway sounds (crowing, snoring, stridor)

11.1.1.7 Presence of excessive secretions, blood, vomitus, or foreign objects in the airway

11.1.1.8 Presence of epigastric sounds

11.1.1.9 Presence of retractions

11.1.1.10 Presence of nasal flaring

11.1.2 Physiologic variables—Repeated assessment of physiologic data by trained professionals supplements clinical assessment in managing patients with airway difficulties. Monitoring devices should be available, accessible, functional, and periodically evaluated for function. These data include but are not limited to:[142,175]

11.1.2.1 Ventilatory frequency, tidal volume, and airway pressure

11.1.2.2 Presence of CO_2 in exhaled gas

11.1.2.3 Heart rate and rhythm

11.1.2.4 Pulse oximetry

11.1.2.5 Arterial blood gas values

11.1.2.6 Chest radiograph

11.2 Endotracheal tube position—Regardless of the method of ventilation used, the most important consideration is detection of esophageal intubation.

11.2.1 Tracheal intubation is suggested but may not be confirmed by

11.2.1.1 bilateral breath sounds over the chest, symmetrical chest movement, and absence of ventilation sounds over the epigastrium;[174,175,177]

11.2.1.2 presence of condensate inside the tube, corresponding with exhalation;[174,176,177]

11.2.1.3 visualization of the tip of the tube passing through the vocal cords;

11.2.1.4 Esophageal detector devices may be useful in differentiating esophageal from tracheal intubation.[178,179]

11.2.2 Tracheal intubation is confirmed by detection of CO_2 in the exhaled gas,[180-182] although cases of *transient* CO_2 excretion from the stomach have been reported.[183]

11.2.3 Tracheal intubation is confirmed by endoscopic visualization of the carina or tracheal rings through the tube.

11.2.4 The position of the endotracheal tube (ie, depth of insertion) should be appropriate on chest radiograph.

11.3 Airway Management Process—a properly managed airway may improve patient outcome. Continuous evaluation of the process will identify components needing improvement. These include response time, equipment function, equipment availability, practitioner performance, complication rate, and patient survival and functional status.

MAE 12.0 FREQUENCY/AVAILABILITY/ DURATION:

Because the need for management of airway emergencies occurs unpredictably, personnel need to be able to respond with the appropriate equipment within 3 minutes, 24 hours/day, 7 days/week.[184] Additionally, a person capable of airway management in the infant should be present at every delivery. A Level-II practitioner should be present at every high-risk delivery.

MAE 13.0 INFECTION CONTROL:

13.1 Implement Universal Precautions including mouth-to-barrier devices and recommendations related to avoidance of the transmission of tuberculosis and other airborne diseases.[185,186]

13.2 Observe all infection control guidelines posted for the patient.

13.3 Disinfect all equipment to be reused on other patients.

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AARC Clinical Practice Guideline

Infant/Toddler Pulmonary Function Tests

ITPFT 1.0 PROCEDURE:

Infant/toddler pulmonary function tests (ITPFTs)

ITPFT 2.0 DESCRIPTION/DEFINITION:

2.1 Infant/toddler pulmonary function tests measure a variety of pulmonary variables in subjects who are generally too young to perform, comprehend, or comply with necessary instructions for conventional pulmonary diagnostic procedures (eg, forced vital capacity, slow vital capacity, panting airways mechanics).

2.2 Subjects are typically tested while sleeping or sedated, may have an artificial airway in place, and may be either spontaneously breathing or mechanically ventilated.

2.2.1 Sedation is required in instances in which the subject's agitation or movements make it difficult to acquire meaningful information.[1]

2.3 ITPFTs may include measurements of:[2,3] static and dynamic pulmonary mechanics (total respiratory system compliance [C_{rs}] and lung compliance [C_L], total respiratory system resistance [R_{rs}] and airway resistance [R_{aw}]), tidal-breathing flow-volume loops, gas dilution/elimination FRC (FRC_{He} and FRC_{N2}), plethysmographic FRC (FRC_{pleth}), rapid thoracoabdominal compression ("squeeze"/"hug") technique for partial expiratory flow-volume curves, and the rapid-deflation technique for expiratory flow-volume curves.

2.3.1 Although these procedures are intended primarily for neonate and infant evaluation, some older subjects may be successfully evaluated, if appropriately sized equipment is employed and methodology limitations are well understood.

2.3.2 Newer techniques, such as methods incorporating inflation of the subject to total lung capacity (TLC) prior to hugging, show promise in assuring maximal maneuvers and obtaining consistent vital capacity (VC) information.[4-6] Recent findings employing oscillation, while simultaneously recording esophageal pressures, provide evidence that flow limitation is being reached. Additionally, use of the interrupter technique to assess airway obstruction is being evaluated.[7] However, because these techniques are currently (1995) in the investigational stages of their development, they will not be addressed in this guideline.

ITPFT 3.0 SETTING:

Testing may be performed, in a variety of settings including, but not limited to hospital laboratories, intensive and intermediate care units, physician offices, and clinics provided infants undergoing conscious sedation can be appropriately monitored in the specific setting.[1,9] The testing site should be adapted to assure the subject's safety during testing.[1]

ITPFT 4.0 INDICATIONS:

ITPFTs may be indicated

4.1 to aid in diagnosis[9] of lung disorders (eg, reactive airways disease);

4.2 to follow the natural history of lung growth, or diseases presenting in infancy[10,11] (eg, cystic fibrosis);

4.3 to evaluate therapeutic responses[12,13] (eg, to medication or physical or respiratory interventions);

4.4 to allow for prediction of risk of subsequent pulmonary dysfunction based upon initial testing.[14]

ITPFT 5.0 CONTRAINDICATIONS:

5.1 Although absolute contraindications to performing ITPFTs have not been described in the medical literature, prior to initiating ITPFTs the technologist should evaluate the patient. Clinical judgment and/or caution should be exercised if the client presents with

5.1.1 active pulmonary bleeding,

5.1.2 open chest wound,

5.1.3 untreated pneumothorax.

5.2 ITPFTs are relatively contraindicated when:

5.2.1 Temporary interruption of ventilatory support will worsen the patient's medical condition and/or pulmonary compliance beyond the benefit of the information gained from performing ITPFTs.

5.2.2 Sedation effects[8] might result in untoward clinical events in patients with cardiopulmonary and/or neurologic disease presenting with conditions for which sedation is contraindicated unless airway patency/protection is assured and maintained.

5.2.2.1 central hypoventilation;

5.2.2.2 pre-existing central nervous system depression;

5.2.2.3 obstructive sleep apnea;

or in patients in whom one or more of the following is present:

5.2.2.4 severe obesity;

5.2.2.5 esophagitis or gastritis;

5.2.2.6 hepatic or renal dysfunction;

5.2.3 The level of patient sedation/paralysis is inadequate;

5.2.4 The patient is uncooperative or combative.

ITPFT 6.0 PRECAUTIONS/HAZARDS AND/OR COMPLICATIONS:

Although ITPFTs are generally safe procedures, the following untoward events may occur:

6.1 vomiting with aspiration with consequent apnea and laryngospasm and/or bronchospasm (The forced deflation technique requires tracheal intubation.[15])

6.2 pneumothorax

6.3 increased intracranial pressure

6.4 loss of airway patency

6.5 transmission of contagion via improperly cleaned equipment or as a consequence of the inadvertent spread of droplet nuclei or body fluids (patient-to-patient or patient-to-technologist)

6.6 oxygen desaturation due to

6.6.1 a worsening of ventilation-to-perfusion mismatch and hypoventilation as a consequence of sedation and/or positioning;

6.6.2 interruption of oxygen therapy or failure to preoxygenate the patient prior to performing the forced deflation technique;

6.6.3 temporary loss of distending pressure.

ITPFT 7.0 LIMITATIONS OF METHODOLOGY/ VALIDATION OF RESULTS:

Large intra- and interlaboratory differences in measured ITPFTs and in percent of predicted values have been observed. These differences are attributed to variations in patient preparation, testing techniques, equipment, computational algorithms, and errors in gas analysis.[1] The choice of equipment, equipment calibration, and tester training may also affect the measured values. Technique standardization and validation is required.[1]

7.1 Typical methods used for ITPFTs and their limitations are[15,16]

7.1.1 Rapid thoracoabdominal compression (RTC) squeeze/hug technique.

7.1.1.1 Increased upper airway resistance may interfere with the accuracy of intrathoracically determined flows.

7.1.1.2 Air flow may be affected by upper airway obstruction and nasal compression or by head and neck positioning. (Subject positioning must minimize pharyngeal narrowing.)

7.1.1.3 Flows should be referenced to lung volumes.[17]

7.1.1.4 Reflex glottic closure (complete or partial) may limit flow.

7.1.1.5 Variations in end-expiratory levels, or functional residual capacity (FRC), may affect measurements 'at FRC' making therapeutic evaluations (eg, efficacy of bronchodilator or surfactant) difficult.

7.1.1.5.1 Sleep state and subject positioning affect FRC.[1,15]

7.1.1.5.2 Repeated hugs alter true FRC. Rest intervals must be provided between serial measurements.

7.1.1.6 Unless actual flow limitation (defined as no further increase in flow despite increasing pressure) is reached, intra- and intersubject comparisons are problematic.

7.1.1.6.1 Whether flow limitation can actually be reached (using this technique) and the best method for determining flow limitation is controversial.

7.1.1.6.2 Hug pressures may range ≤ 40 to > 80 cm H_2O.[17] However, if too little pressure is transmitted to the pleural space, flow limitation will not be achieved. Conversely, excessive pressures may alter the shape of the curve, via negative effort dependence.[17,18]

7.1.1.7 The flow-volume relationship produced by the RTC technique represents a small portion of the entire MEFV curve.[19] (Inspiratory efforts during squeeze may reduce expiratory volumes and/or flows.)

7.1.1.8 Improperly sized and positioned 'hug' bag.[17,20,21]

7.1.1.9 Pneumotachometer with inappropriate flow range.

7.1.2 Compliance and resistance measurements obtained by various techniques may be difficult to interpret,[22] and further work is required to define their validity in normal infants and those with lung disease. For example, assumptions regarding linearity of lung compliance and resistance may not necessarily be valid with disease states.

7.1.2.1 Evaluation of lung compliance requires placement of an esophageal balloon to measure transpulmonary pressure.

7.1.2.1.1 Inappropriate balloon placement and other factors (such as distortion due to paradoxical chest-wall movement) invalidate transpulmonary pressure measurements.

7.1.2.1.2 The instability/flexibility of the premature infant's chest wall may invalidate results.

7.1.2.2 Evaluation of respiratory system compliance is not valid unless ventilatory muscles are relaxed (ie, paralyzed, highly sedated, or demonstrating effective Hering-Breuer reflex).

7.1.2.2.1 Effects of sleep deprivation, sedation, anesthetics, and muscle relaxants may alter FRC, bronchomotor tone, and upper airway resistance.

7.1.2.2.2 Pneumotachometers and occlusion valves increase system dead space and resistance, which may alter tidal volume, respiratory rate, and FRC.

7.1.2.2.3 Leaks around artificial airways and masks lead to erroneous measurements.

7.1.2.2.4 Single breath occlusions should be referenced to lung volume (due to changing FRC). Multiple occlusion procedures provides an average expiratory compliance over a range of tidal volumes.

7.1.2.3 Measurements of R_{aw} and R_{rs} may not be equivalent in subjects having either upper or lower airway obstruction.[23]

7.1.3 Limitations of gas dilution/elimination methods (eg, helium-dilution or multiple-breath nitrogen washout [MBNW] techniques, FRC_{He} and FRC_{N2}, respectively) to measure FRC include

7.1.3.1 system dead-space must be precisely sized to the patient being studied;

7.1.3.2 MBNW results are questionable with $F_{IO2} > 0.7$ to 0.8;[15]

7.1.3.3 background gas flow must exceed the subject's peak inspiratory flow;

7.1.3.4 leaks invalidate measurements (tight mask seals, cuffed ET tubes, or cricoid pressure should be incorporated);

7.1.3.5 analyzer response time, lag time between measuring flow and gas

concentration, and sampling rate affect results;

7.1.3.6 the number of tests performed and test variability.

7.1.3.6.1 Agreement on test reproducibility criteria (in the clinical arena) and the length of time required to wait between sequential FRC measurements is currently lacking (1995).[24]

7.1.3.6.1.1 Recommendations based on clinical experience include performance of at least 6 FRC measurements, discarding the highest and lowest values and reporting the average value of the 4 to 5 FRC_{He} or FRC_{N2} measurements, that have a coefficient of variation of ≤ 8%. (See also **9.0 Assessment of Outcome/Test Quality**)

7.1.3.6.1.1.1 Because infants with lung disease frequently have a high degree of variability in their FRC measurements, reproducibility criteria should exceed those published for adults[19] (reporting the average of at least two FRC_{He} values that are within 10% of each other[15])

7.1.3.6.1.1.2 The number of sequential FRC_{N2} tests (ie, 3 to 5) and corresponding mean coefficient of variation (CV) between tests (3.9% [range = 0.2-9.0%; subjects = 50] to 11.9% [range = 2.8 25.3%; subjects = 25]) have been described in the research setting.[25,26] Reproducibility of multiple FRC_{He} measurements in healthy infants has been reported with a mean CV ± SD of 4 ± 2.8%.[27]

7.1.3.6.1.2 In the clinical setting, a reasonable waiting period, between sequential measurements, may be equal to the time it took to perform the last FRC measurement

7.1.3.6.1.2.1 A 5-minute wait interval, between consecutive FRC_{N2} measurements, has been reported[26] in a research study.

7.1.3.6.1.3 To ensure testing continuity, institutional ITPFT policies and procedures must contain statements about the validation methodologies used for determining and reporting FRC values.

7.1.3.7 Recommendations are lacking for determining equilibration criteria.

7.1.3.8 Technique underestimates FRC when compared to FRC_{pleth}.[25,28]

7.1.3.9 Sleep stage[29] and sedation may affect FRC level.

7.1.3.10 Timing and speed of valve closure for switch-in/switch-out time should be standardized. The proficiency of the technologist for effectively switching-in at FRC is critical because the derived value is dependent upon this ability.

7.1.4 Tidal breathing flow-volume loops may provide limited information if used alone.[30]

7.1.5 Forced deflation technique:

7.1.5.1 Procedure is suitable only for patients with an artificial airway,[9] but leaks around the endotracheal tube cuff results in inaccurate measurements

7.1.5.2 Pneumotachometers must be calibrated according to the gas densities encountered during testing.

7.1.5.3 Endotracheal tube size may alter flows.

7.1.5.4 The effects of altering volume history on lung function needs to be better delineated.

7.1.5.5 Patients need to be paralyzed or deeply sedated.

7.1.5.5.1 Sedation levels may affect FRC.

7.1.5.5.2 Pharmacologic agents (anesthetics, muscle relaxants) may affect testing (ie, alter bronchomotor tone).

7.2 Little has been published on ITPFT predicted values or reference equations. The choice of

reference equations may affect the final interpretation of measured values.

7.3 Validation of the testing technique/equipment may include but is not limited to:

7.3.1 Accuracy of the volume or flow-measuring device and ability to maintain accuracy with varying gas concentration. The accuracy of the calibrating syringe's standard volume should be assessed on a regular basis and revalidated annually or any time accuracy is suspect.

7.3.2 Gas analyzers should have a 2-point calibration before each test, and linearity should be formally checked (using a minimum of 3 points) at least every 6 months. Analyzers should be maintained according to manufacturer recommendations.

7.3.3 The creation of predicted reference ranges is difficult in the average laboratory. The recognition that there is little published data cannot be overemphasized at this point in the evolution of the ITPFT field. 'Normal' standard subjects may be used to establish an acceptable intrasubject coefficient of variation[31] and serve as a quality control population.

ITPFT 8.0 ASSESSMENT OF NEED:

Determination that valid indications are present.

ITPFT 9.0 ASSESSMENT OF OUTCOME/ TEST QUALITY:

Outcome and test quality are determined by ascertaining that the desired information has been generated for the specific indication(s) and that validity and reproducibility have been assured. Each laboratory should standardize procedures and demonstrate intertechnician reliability.[1] Test results can only be considered valid if they are derived according to and conform to established laboratory quality control and quality assurance protocols. These protocols should address test standardization and reproducibility criteria that include the methodology used to derive and report the ITPFTs.

9.1 ITPFTs performed for the listed indications are valid only if the instrumentation functions acceptably and the maneuvers are obtained in an acceptable, reproducible fashion.

9.2 Report of test results should contain a statement by the technician performing the test about test quality and if appropriate, which recommendations were not met.

ITPFT 10.0 RESOURCES:

10.1 Equipment: Equipment specifications should conform to recognized standards (eg, American Thoracic Society spirometry standards[32]) and where applicable, be FDA approved.

10.1.1 Distinctive pneumotachograph, helium analyzer (katharometer) and nitrogen analyzer performance specifications. Appropriate use of gas analyzers is dependent upon the methodology employed.

10.1.2 Gases must be medically certified.

10.1.3 Size-appropriate resuscitation equipment (including appropriate pharmacologic agents) must be readily available.[1]

10.1.4 Sedation monitoring equipment must be available (eg, continous pulse oximetry with pulse rate).[1]

10.2 Personnel:

10.2.1 ITPFTs should be performed under the direction of a physician trained in infant pulmonary function testing methodologies (including limitations and applications). The value of ITPFT results are compromised when a test is administered and/or interpreted by inadequately trained personnel.

10.2.2 Testing personnel should be specifically trained (with verifiable training and demonstrated competency) in all aspects of ITPFTs, including equipment theory of operation, quality control, and test outcomes relative to diagnosis and/or medical history. Proficiency must also be demonstrated relative to their ability to calibrate equipment, apply ancillary devices to the client, perform the test, monitor the patient and determine the quality of the test.

10.2.2.1 Testing personnel should also be trained in basic life support.[1]

10.2.2.2 At least one of the following credentials is recommended: CRTT,

RRT, CPFT, RPFT, LPN, RN, MD, DO.

ITPFT 11.0 MONITORING: (Also see Section 9.0 ASSESSMENT OF TEST QUALITY)

The following should be monitored during ITPFT determinations

11.1 Test data of repeated efforts (ie, reproducibility of results) to ascertain the validity of the results (The final report should contain a statement about testing conditions and test quality.)

11.2 The final report should contain the requested parameters and lung-volume corrected values (if applicable).

11.3 The patient for any adverse effects of testing

11.3.1 Infants undergoing conscious sedation should be appropriately monitored,[8] with sedative information included in the final report.

11.3.2 Patients on supplemental oxygen may require periods of time to rest (on oxygen) between trials.

ITPFT 12.0 FREQUENCY:

The frequency at which ITPFT measurements are repeated depends on the clinical status of the patient and the indications for performing the test.

ITPFT 13.0 INFECTION CONTROL:

Infant/toddler pulmonary function tests are relatively safe procedures, but the possibility of cross-contamination exists, either from the patient-patient or patient-technologist interface.

13.1 Universal Precautions (as published by the Centers for Disease Control) should be applied in all instances in which there is evidence of contamination with blood (eg, pneumotachometers and adapters). Although Universal Precautions do not apply to saliva or mucus unless it contains blood, other potentially hazardous organisms may be present in these fluids even in the absence of blood, and the appropriate use of barriers and hand washing are recommended.[33,34]

13.2 Due to the nature of some ITPFT maneuvers and the possibility of coughing when the test is performed by subjects with active infection with *M tuberculosis* or other airborne organisms, the following precautions are recommended:[35]

13.2.1 If a maneuver is likely to stimulate or induce a cough, disposable gloves, protective outerwear, along with masks (which comply with OSHA requirements) and protective eyewear should be utilized. This personal protective equipment is also to be used when testing patients with known or suspected, potentially infectious airborne disease(s).

13.2.2 The room in which ITPFTs are performed should meet or exceed the recommendations of U.S. Public Health Service for air changes and ventilation. The most desirable arrangement may be to maintain a specially ventilated area in the testing department for isolation patients.

13.3 Any parts of the system that come into contact with the subject should be disposable or sterilized between patients. If sterilization is not feasible, then high-level disinfection should be performed.[36] All cleaning should comply with manufacturer recommendations. Several pneumotachometers and/or valving assemblies may be required if cleaning cannot be performed in a timely manner between patients.[1]

13.4 The use of bacterial filters is controversial.[36]

13.4.1 Attachment may result in added system dead space and may invalidate pneumotachometer accuracy by increasing total system resistance of the apparatus.

13.4.2 Filter resistance should be subtracted from R_{aw} (and related parameters)

13.4.3 If filters are used in gas-dilution procedures, their volume should be subtracted when FRC is calculated.[24]

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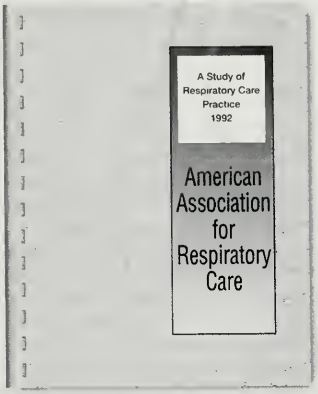

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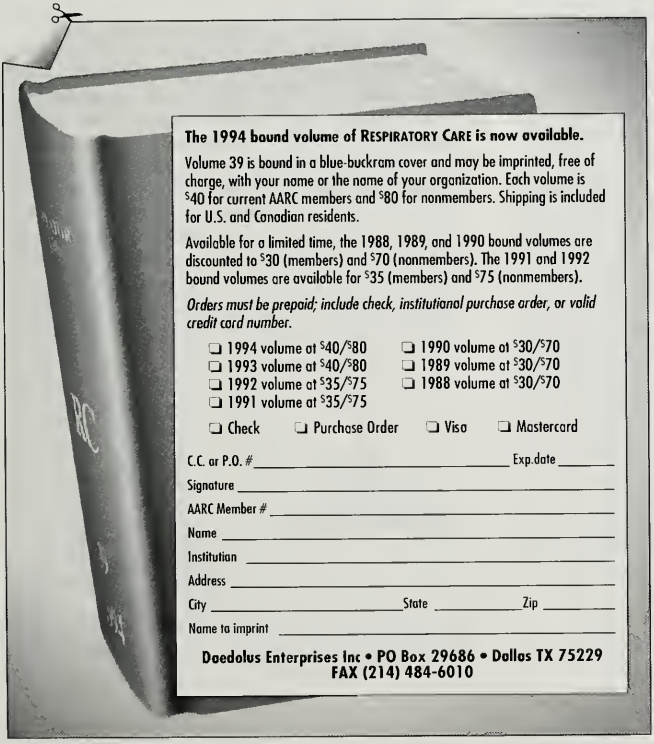

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RETROSPECTROSCOPE

Whodunit (And Why)[1]

Part II*

In Part I of "Whodunit (and Why)" (*American Review of Respiratory Disease*, June 1976), I asked the question "How many of these key studies [crucial to clinical advances in cardiovascular and pulmonary medicine and surgery] were done to solve a specific clinical problem related to the prevention, diagnosis or treatment of a specific [human] disease; how many were done simply to learn something, completely unrelated to clinical medicine or surgery or to the genesis or course of a disease?" And because the number of such studies was very large (41% of all key articles), I believed it important to list some of these for you. Part II continues this annotated list.

Blood pumps. Everyone knows that a pump is an essential component of a heart-lung machine. Galletti and Brecher (20) devote three chapters of their *Heart-Lung Bypass* to pumps. One of the early blood pumps was the roller pump that DeBakey used in 1934 for transfusing blood—obviously a clinically oriented effort (21). However, two years before DeBakey, Van Allen described a similar pump in *JAMA* and even then disclaimed any originality for his device (22). And four years before Van Allen, two English physiologists, Bayliss and Müller, described the same roller pump, which they turned over to C. F. Palmer, Ltd., for manufacture (23). Bayliss was a cardiovascular physiologist who used his pump for laboratory experiments and not for transfusions in man. Curiously, there are no references in any of these three papers to the earlier papers in the series or to Issekutz, who used a roller pump to perfuse the liver of animals in 1927, or to Kelly, who received U.S. Patent 314851 for one in 1885!

The Potts clamp for aortapulmonary artery anastomosis. In the early days of the Blalock-Taussig "blue baby" operation, which created an end-to-side shunt between a branch of the aorta (such as the subclavian) and the right or left pulmonary artery, Potts devised a clamp that permitted direct side-to-side anastomosis between the aorta and pulmonary artery without sacrificing a subclavian artery. The key to the Potts operation was permitting blood flow to continue through both vessels while they were being joined; this required devising a new clamp that closed one side of the aorta (where the anastomosis was to be performed) but left an open channel for blood flow through the other side (24). Definitely a clinically oriented effort, to solve a specific surgical problem. But in the same journal (*JAMA*), only 36 years earlier, one of America's leading physiologists, G. N. Stewart (of indicator-dilution fame) described the same device "to clamp off temporarily a longitudinal portion at one side of a blood vessel while the circulation is allowed to go on through the remainder of the lumen" (25) (Fig. 4). Although he was a physiologist who devised the clamp for physiologic experiments (such as partial occlusion of an artery), he speculated on its possible clinical uses: to arrest hemorrhage from a wound in a large vessel while suturing it; to perform lateral anastomoses of blood vessels when it is undesirable to occlude completely either vessel (the 1946 Potts operations!); to implant one vessel laterally onto another. Photographs of Stew-

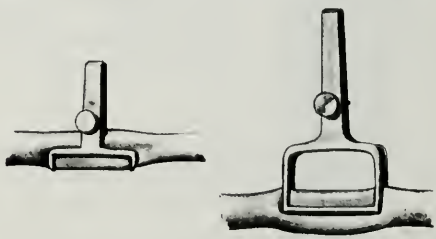

Fig. 4. Two clamps devised by G. N. Stewart in 1910 to occlude part of an artery (to be operated on) while blood flow continued through an open channel (26,27).

[1] Studies on which this article is based were supported by Contract 1-HO-1-2327 from the National Heart and Lung Institute, and by grants from the Commonwealth Fund and the Burroughs-Wellcome Fund.

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art's clamp appeared in the two leading texts of experimental surgery of that era: Guthrie's in English in 1912 (26) and Jeger's in German in 1913 (27). A recurring phenomenon in many papers recording the great cardiovascular surgical triumphs of the 1940s and 1950s was the failure to mention previous work in the field. Was this piracy, or disinclination to use the library? And which was worse?

Preservation of blood and tissues. Carrel preserved arteries and veins for future use as grafts in his experimental work in 1912 (28). In 1919 Guthrie preserved vessels in formaldehyde and found that they served as scaffolding for the ingrowth of a new lining (29). Rous and Turner developed a method for preserving red blood cells in 1916 (30), although preserved blood appears not to have found its way to World War I battlefields in 1917 or 1918; for the most part, physicians used 6 per cent gum arabic in 0.9 per cent sodium chloride.

A fascinating discovery of the early 1900s was freeze drying of tissues by Shackell (31) while he was a chemist working in the Missouri Agricultural Experiment Station. His initial goal was to develop a quick and precise method to measure water content of various materials such as soil, cornchop, feces, milk, honey, levulose, butter, cheese, vinegar, and soap. This he accomplished by an improved method of vacuum desiccation. He then turned his attention to measuring the concentration of liver glycogen and found his values varied from day to day because of rapid postmortem hydrolysis of glycogen. How could he maintain premortem concentrations while drying tissue post mortem? The answer was to freeze the liver first and then subject it to vacuum desiccation.

Shackell then moved from the Agricultural Station to the Department of Physiology of the University of Missouri's School of Medicine and soon realized that his freeze-dry method had medical applications. He found that complement was preserved in freeze-dried guinea pig serum, that the coagulation factors remained intact in freeze-dried dog's blood, and that the fixed virus of rabies survived freeze-drying of rabbit brains.

In 1935, Elser, Thomas and Steffen (32) improved on Shackell's technique and used it to preserve antisera and antigens. During World War II, Flosdorf and associates first freeze-dried human plasma on a large scale and then did the same for penicillin (33,34); thus, they solved the two urgent problems of how to store plasma for wartime needs and how to preserve penicillin, notoriously unstable in solution. Since then, freeze drying has had many medical applications and even more industrial uses (e.g., the burgeoning business of freeze-dried coffee).

Cathode ray oscilloscope. This is now standard equipment in intensive care units for monitoring blood pressure, the electrocardiogram and electroencephalogram, end-tidal CO_2 tension, and other cardiovascular and respiratory variables. The original "cathode ray indicator tube" was devised in 1897 by Braun for the visual study of oscillatory and transient phenomena in electric circuits; for this achievement, Braun received the 1909 Nobel Prize in Physics (jointly with Marconi). The first biologic use of the cathode ray oscilloscope was in the field of basic neurophysiology. Gasser and Erlanger (35) were trying to record the true form of the action potential in nerve fibers but found that the string galvanometer was too sluggish. They needed an inertia-free system and found it in 1922 in Western Electric's modified Braun tube; this was a gas-focused cathode ray tube that operated on low voltages with simple auxiliary circuits. The Western Electric Company refused to sell their tube to Gasser and Erlanger, presumably because the company feared the possibility of infringement of its patents. So the neurophysiologists simply made their own using a distillation flask (36). The cathode ray oscilloscope became indispensable in neurophysiologic studies, came into widespread laboratory use as a monitoring device, and eventually "went clinical."

Blood oxygen tension. The name of Leland Clark is rightly associated with the development of the oxygen electrode, but several important basic investigations preceded his work. One was Danneel's finding in 1897 that, during the electrolysis of water using platinum electrodes, there was an approximate proportionality between the current and the concentration of dissolved O_2 when the e.m.f. applied was 0.02 volt. Another came 25 years later, when Professor Kučera at Prague suggested to his young associate, Jaroslav Heyrovský, that he should study certain irregularities in connection with the capillarity of mercury. This led to the invention of the polarograph (37) and to a Nobel Prize in Chemistry. In presenting the Prize to Heyrovský, Professor Ölander said:

> This [the study of irregularities in the capillarity of mercury] was one of the innumerable small problems constituting science. Heyrovský let the mercury flow through a glass capillary and weighed the drops. It was a slow and tedious method, and he resolved instead to measure the electric current obtained when he put a tension [e.m.f.] between the mercury in the capillary and that collecting at the bottom. The glass capillary does not terminate in the air, but in a solution, through which the current now will flow.
>
> Heyrovský found that this device could be used for something much more important than the original problem. It could be used for ascertaining very small quantities of the most diverse substances dissolved in the water, and moreover, to measure their percentages.
>
> Important new discoveries are found where they are not expected.... Discoveries are made by some scientist who noticed something strange, possibly by two [scientists], independently and in different countries. Then it is important that future team leaders and authorities granting funds do not keep him too strictly to attend his job, but give him a chance to pick up the unexpected new things, in spite of the chance of finding them being diminutive (38).

The polarograph could measure, among other things, the concentration of oxygen dissolved in liquids and it stimulated interest in measurement of oxygen tension in tissues. The first use of the platinum electrode to measure oxygen tension in animal tissues appears to be that of Davies and Brink (39). Clinically oriented? No, Davies and Brink were biophysicists interested in measuring the oxygen consumption of resting and active nerve tissue. When they placed nerve tissue in a small, saline-filled, closed chamber, they could run saline slowly through the chamber and measure the oxygen tension in the inflowing and outflowing fluid; from these values, they calculated the oxygen removed by tissue. They also used a platinum electrode to measure the oxygen tension of the surface of the brain. However, during research in aviation physiology, they decided to put a platinum electrode in the plunger of a syringe and measure the oxygen tension of blood. They ran into the problem that whole blood "poisoned" the bare tip of the wire; to protect the tip they covered it with a collodion membrane that allowed oxygen, but not blood or plasma, to diffuse to the platinum electrode (see their "Note added in proof," p. 533 [39]). In 1950 Brink wrote, "The component parts exist for the successful development of an electrical instrument for the measurement of dissolved oxygen in whole blood *in situ*. All that is required is an intensive program of development of the electrode for this specific purpose. Those interested in using the measurements must decide whether such a development is justified" (40).

In 1953, Clark decided. He had devised the first successful bubble oxygenator for use in cardiopulmonary bypass operations. To test its efficiency, he needed to know continuously the oxygen tension of blood after passage through the oxygenator, and for this he had to develop the oxygen electrode and make it dependable and accurate (41).

Chromatography. One of the most powerful and versatile analytic methods used in clinical medicine is chromatography. But its first use was in pure science. Michael Tswett, a Russian botanist, devised it as a way to separate and identify the constituents of green leaves; he extracted them with petrol ether and poured the mixture over a vertical column of calcium carbonate. The main constituents, in this case pigments (green chlorophyll and yellow carotenoids), migrated at different rates and separated as distinct color bands; hence the term chromatogram. In 1906, Tswett wrote:

> Like light rays in the spectrum, so the different components of a pigment mixture are resolved on the calcium carbonate column according to a law and can be estimated on it qualitatively and also quantitatively. Such a preparation I term a chromatogram and the corresponding method, the chromatographic method.
>
> It is self-evident that the adsorption phenomena described are not restricted to the chlorophyll pigments, and one must assume that all kinds of colored and colorless chemical compounds are subject to the same laws I have so far investigated lecithin, alkannin, prodigiosin, sudan, cyanin, solanorubin, and acid derivatives of the chlorophyllins with positive success (42).

The term chromatogram is no longer wholly appropriate because it is used to separate colorless compounds (as Tswett predicted) and gases, but it is useful to remind us of its origin—the study of the pigments in green leaves.

Beta receptors. From our knowledge of β-receptors (properly termed β-adrenoreceptors) have come many clinically useful drugs, those that stimulate (e.g., isoproterenol) and those that block (e.g., propranolol) these receptors. How did the idea of β-receptors come about. First there had to be the notion that drugs acted on specific parts of a cell (receptor substances) and not on the cell as a whole. Then there had to be the concept and experimental proof of humoral transmission of nerve impulses, that nerve impulses did not leap as sparks from preganglionic fibers to ganglion cells or from postganglionic fibers to muscle or glands, but continued across anatomic gaps by liberation of highly specific chemical substances (acetylcholine or norepinephrine), formed locally within nerve endings. Walter Cannon was pretty sure in 1937 that epinephrine came in two forms, E for excitatory and I for inhibitory; the evidence was based in part on the effect of blocking agents. For example, Dale knew in 1906 that ergotoxine blocked some of the actions of epinephrine but left others intact; ergotoxine not only blocked the characteristic increase in blood pressure from an injection of epinephrine, but reversed it (epinephrine, given after ergotoxine, actually lowered blood pressure).

In 1948, Ahlquist realized that the classification of epinephrine effects into excitatory and inhibitory was holding up progress in this field (43). After all, norepinephrine was "excitatory" because it constricted smooth muscle of systemic arterioles, but it also inhibited smooth muscle in the gut: isoproterenol was "inhibitory" because it dilated blood vessels and bronchioles, but it also excited the heart. He therefore proposed that there were two types of adrenoreceptors—alpha and beta—and that drugs be classified on the basis of whether they stimulated or blocked α- or β-preceptors (exclusively, strongly, weakly, or not at all). Dibenamine (never useful clinically) served to block α-receptors and dichloro-isoproterenol (also never useful clinically) served to block β-receptors. The stage was now set for a meaningful and systematic search for highly specific stimulant and blocking agents and soon important new drugs were synthesized (e.g., propranolol).

To date, six scientists who have worked in this field have received Nobel Prizes: Dale and Loewi in 1936, Katz, von Euler, and Axelrod in 1970, and Sutherland in 1971, all for basic research that eventually led to important clinical advances in therapeutics.

Dicumarol. The story of the first oral anticoagulant began in 1922 when Schofield reported on a new disease that occurred exclusively in cattle fed on mouldy sweet clover hay, and was characterized by subcutaneous swellings caused by

hemorrhages, followed by sudden deaths. In all cases there was acute anemia and a greatly diminished amount of plasma prothrombin (44). (Editors were a different breed in 1922 than now. Schofield apologized at the end of his paper, "This paper has been written very hurriedly as the editor was insistent that it appear in this issue. I therefore ask the readers to be lenient in their criticism, not of the contents, but of the arrangement of the material presented.")

The next chapter in the story was by Roderick of the North Dakota Agricultural Experiment Station. He confirmed that the disease was associated with progressive decrease in plasma prothrombin but found no pathologic evidence of serious liver damage (understandable now that we know that the hepatic lesion is a specific one limited to prothrombin formation) (45).

In 1940-41, Campbell and Link (46), also working in an Agricultural Experimental Station, in Wisconsin, decided to extract and concentrate material from spoiled sweet clover hay; they soon isolated dicumarol in a pure state ($C_{19}H_{12}O_6$). From this point on, dicumarol moved in two directions: (1) One of its congeners, warfarin, was used deliberately to kill animals—this time rodents instead of cattle—but by the same mechanism (decrease in prothrombin concentration, spontaneous hemorrhages, and death); it had a huge market as a rodenticide. (The name "warfarin" was not derived from "warfare on rats" but from *W*isconsin *A*lumni *R*esearch *F*oundation, which received profits from the patents.) (2) As dicumarol, it entered clinical medicine after some of Link's biochemists tried it out on themselves and concluded that "in view of the prothrombin-reducing or inactivating properties of this dicoumarin, the spread between the detectable and lethal dose, together with the relative ease with which it may be prepared and administered, it would appear that its anticoagulant properties merit consideration by the physiologist and hematologist."

Para-aminosalicylic acid (PAS) therapy of pulmonary tuberculosis In 1940, Bernheim wrote a note to *Science* on the metabolism of tubercle bacilli (47). These bacilli, when placed in the Warburg apparatus, did not readily oxidize substances such as carbohydrates, amino acids, or hydroxy acids, but Bernheim found that when he fed them sodium salicylate, their uptake of O_2 more than doubled. Because the increase in O_2 uptake was proportional to the concentration of salicylate, Bernheim suggested that the bacilli oxidized salicylate as a substrate and that possibly it was one of their normal metabolites.

This observation might have gone unnoticed except that in 1931 Quastel and Wheatley (48) had discovered the principle of "competitive inhibition" when they demonstrated that malonic acid inhibited the oxidation of succinic acid by bacteria, brain and muscle, and in 1940 Woods (49) had applied this principle to explain the antibacterial action of sulfanilamide (that para-aminobenzene-sulfanilamide competed with an essential bacterial metabolite, para-aminobenzoic acid, and so prevented growth of streptococci.) Because salicylate is ortho-hydroxybenzoate, Bernheim's observation, coupled with Wood's findings, might be important for tubercle bacilli.

In 1946, Lehmann followed up Bernheim's work but found no increase in oxygen consumption when salicylates were added to tubercle bacilli. Lehmann might have given up at this point but suddenly realized that he had been using nonpathogenic tubercle bacilli and Bernheim had worked with pathogenic strains. Lehmann then found an essential metabolic difference between pathogenic and nonpathogenic tubercle bacilli: The former showed a positive salicylate effect, the latter did not, (50). Lehmann stated, "the differences found seem to open up new pathways for studying the pathogenicity of the tubercle bacillus." He really meant *old* pathways because he went the competitive-inhibition route of Quastel and Wheatley (48) and Woods (49); he studied more than 50 derivatives of salicylates to learn which might inhibit growth of tubercle bacilli, presumably by fooling the bacilli to accept it instead of salicylate and so slowing or halting bacterial growth. He found PAS to be the most effective inhibitor; so began chemotherapy for tuberculosis.

Anecdotes?

In this two-part Retrospectroscope I've related how 42 discoveries that had no relevance to clinical medicine at the time they were made have been important, or essential, to 24 important advances in clinical medicine and surgery. Some of the nonclinically oriented discoveries have come from physics, chemistry, botany, agriculture, microbiology, physiology, pharmacology, and biochemistry; some have been made by a research surgeon, a veterinarian, a photographer, even by a clergyman. Some have led to important new diagnostic tests, some to whole new fields of surgery, some to new drugs, including chemo-therapeutic agents.

At the beginning of Part 1 (51), I said that our analysis of 529 key articles showed that 41 per cent (217) reported work that, at the time it was done, had no relation whatever to the disease it later helped to prevent, diagnose, treat, or alleviate. I also said that it was time for scientists to stop the anecdotal, "let-me-give-you-an-example" approach to influencing national biomedical science policy. By recounting for you only 42 of the 217 key articles classified as "not clinically oriented," have I slipped into the anecdotage that I have criticized in others? I think not; 42 examples, coming from diverse fields, should be enough to convince you of the importance of supporting nondirected research (research not clinically oriented).

If the Editor will give me five times the space, I'll gladly tell you about the other 175 examples. But other matters deserve "equal time," such as the role of purely clinical observations, of engineering, and of development in advancing medicine and surgery—and learning how often clinical observations led to important new research in basic science that in turn led to new clinical advances. More of these matters later.

JULIUS H. COMROE, JR.

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Pediatric Respiratory Disease: Diagnosis and Treatment, by Bettina C Hilman, Professor of Pediatrics and Chief, Pulmonary/Allergy, Louisiana State University Medical Center at Shreveport. 919 pages, hardcover, illustrated. Philadelphia: W B Saunders, 1993. $210.

Dr Hilman successfully addresses the needs of the clinician with this comprehensive textbook about pediatric pulmonary medicine. She combines her own experience in pediatric pulmonology, allergy, and immunology with the expertise of 126 pediatric subspecialist colleagues in infectious disease, critical care, radiology, otorhinolaryngology, cardiology, gastroenterology, hematology, pharmacology, neonatology, genetics, and surgery. The text represents the works of many scientists and clinicians who have played key roles in the development of today's pediatric pulmonology.

We found this book to be useful as a reference text for the clinician in the everyday management of pediatric pulmonary conditions in the hospital, clinic, or home. Respiratory therapists, nurses, and physicians involved in general or specialty care can use this readable text to improve their knowledge, assessment, and management skills when treating a child with respiratory problems.

The book's 97 chapters are logically divided into sections for ease of use. The first section addresses general background information on pulmonary physiology, mechanics, defenses, and mucology. It also covers the epidemiology of pediatric pulmonary disease and the emergence of self-management programs in the long-term care of the child.

The second section focuses on the evaluation of the patient with respiratory symptoms. Clinical and radiologic assessment, breath sounds, diagnostic imaging, noninvasive blood gas monitoring, and pulmonary function testing in infants, children, and adolescents are covered in detail. Special diagnostic techniques, such as rigid and flexible bronchoscopy, bronchoalveolar lavage, bronchial provocation, and rapid diagnostic techniques for viral respiratory infections, are discussed in relation to the performance of the procedure, current indications, contraindications, complications, and reliability. Common pediatric conditions such as recurrent cough, recurrent pneumonia, and chest pain are addressed in separate chapters that offer the clinician complete differential diagnosis and detailed strategies for evaluation.

The third section reviews pediatric respiratory infection and associated conditions including pediatric HIV infection, bronchiolitis, obliterative bronchiolitis, bronchiectasis, epiglottitis, croup, and lung abscess. Bacterial, fungal, viral, mycoplasmic, and chlamydial pneumonia are presented in separate detailed chapters. Additionally, for easy cross-referencing, there are composite chapters on the respiratory sequelae of viral illness and common pneumonias seen with the immunosuppressed child. For reasons we do not understand, "Diseases with a Respiratory Component" is included under this section on Respiratory Infections. These chapters address the pulmonary complications of a variety of cardiovascular, collagen-vascular, interstitial, neurologic, and musculoskeletal disorders, as well as sickle cell anemia.

"Noninfectious Disorders of the Respiratory Tract," the largest section of the book, covers a variety of genetic, environmental, neoplastic, traumatic, and emergency conditions. It also reviews common clinical conditions and management of atelectasis, pulmonary emboli, pulmonary edema, gastroesophageal reflux, hemoptysis, and pneumothorax. This allows the clinician to quickly access possible differential diagnoses when confronted with these symptoms .

Additional sections on asthma, cystic fibrosis, and immunologic disorders are comprehensive and represent current theory, as is expected in a text of pediatric pulmonology. The final section on management of pediatric respiratory problems is timely, offering advice on pharmacology, environmental control, surgical intervention, including transplantation and thoracotomy, oxygen therapy, airway management, and mechanical ventilation.

Duplication in a textbook such as this is unavoidable as the author attempts to meld complete discussions of interrelated topics that have been written by various authors. Repetitive topics included respiratory physiology, pulmonary function, pneumonia, and viral illnesses. This repetition becomes apparent as one reads the entire text. The clinician who uses the book in search of a specific diagnosis, condition, or treatment will find in each chapter the necessary supporting discussion of the desired topic as well as references to other information located elsewhere in the text.

In our opinion, the minor drawbacks of this textbook include the lack of illustrations in chapters such as Croup and Epiglottitis, Immunodeficiency and Lung Disease, Heart-Lung and Lung Transplantation. The airway management chapter did not include a table of appropriate pediatric endotracheal tube sizes. There are a few chapters in the book that did not seem to be applicable to most clinicians, such as the chapter on phonopneumography, and the intense technical review of the genetics of cystic fibrosis. The chapters about mechanical ventilation lacked discussion of permissive hypercapnia. In addition, the discussion of Cardiopulmonary Resuscitation in the Pediatric Patient referenced the 1988 *Textbook of Pediatric Advanced Life Support*, which does not include the current use of high-dose epinephrine. With these exceptions noted, the references cited throughout the text are abundant and reflect current theory.

The highlights of Hilman's book that set it apart from the other bibles of pediatric respiratory disease are Pediatric Human Immunodeficiency Virus Infection, that focuses on the unique pediatric presentation and course of illness; Home Intravenous Antibiotic Use, which addresses the safety, practical, and liability issues for this facet of outpatient management; and Passive Smoking, which is an excellent review of the effects of second-hand smoke in children. The chapter about Chest Pain, written by our distinguished colleague, Dr Martha Franz, is notably excellent. Overall, our opinion is that **Pediatric Respiratory Disease: Diagnosis and Treatment** is well worth the price for any professional who needs quick, detailed information on any aspect of respiratory disease in children.

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The Children's Medical Center
Dayton, Ohio

Hyperactive Software,* Clinical Simulations for use with IBM PCs and Macintosh computers, by Bill Leffler, BA, RRT, 1992. $60 per simulation or set of 13 for $700.

This software series, sold individually or as a set, consists of 13 traditional clinical simulations. Each simulation has information-gathering and decision-making sections, and the student's score is provided at the end. Students have the option of repeating the simulation or going to the answer section where points are assigned to each response.

Status Asthmaticus. In this simulation, a patient with allergic asthma arrives in the emergency room (ER). Based on information gathered by the student, the patient requires pulmonary evaluation and appropriate oxygen, aerosol, or IPPB therapy. The assessment data are adequate for the student to make sound decisions about the need for intubation, initial ventilator settings, troubleshooting ventilator alarms, interpreting blood gas results and making ventilator changes, weaning, extubation, and postextubation therapy.

COPD. In this situation, a COPD patient is seen in the clinic and later in the ER in respiratory distress. Unique to this simulation is a review of pulmonary function results. Although these simulations are intended to prepare the student for national Registry exams rather than pulmonary function boards, I believe the student would benefit more from evaluating numerical values rather than just a description of the values—ie, "...flowrates following bronchodilator administration were significantly improved." The student is also required to recommend bronchial hygiene therapy, suggest a home care plan, and provide limited patient instructions.

Cystic Fibrosis. A patient with cystic fibrosis is seen in the outpatient clinic in this simulation. The student must recommend home bronchial hygiene and instructions for cleaning equipment. The patient is later readmitted for further evaluation and additional recommendations for care. The decisions necessary in this case are not unique but routine in nature giving the student an opportunity to develop the basics of respiratory care.

Chest Trauma I. The student experiences chest-tube management and mechanical ventilation in this scenario of a motor vehicle accident (MVA) patient admitted to the ICU from OR. The case provides a variety of situations for the student including basic patient assessment, ventilator management, extubation, and management of a pulmonary infection.

Chest Trauma II. This MVA patient arrives in the ER, breathing spontaneously and in distress. The student is asked to assess and manage the patient in this situation. Decisions to be made include whether to oxygenate, ventilate, or insert a chest tube. Chest X-ray and chest movement confirm flail chest. Emphasis is placed on ventilator application—initial ventilator settings, changes in settings based on blood-gas results, troubleshooting ventilator alarms, and causes for increasing peak inspiratory pressures. The student is asked to collect data for calculating static lung compliance, but the student is not asked to evaluate the results. I believe the evaluation of collected data is essential for developing a student's thinking skills in the critical care setting. At one point in the simulation, the student must decide to insert a pulmonary artery catheter, fluid challenge, add inflation hold, perform a tracheostomy, or stabilize a flail chest based on normal blood gas values. I believe additional data are needed to make decisions that support stabilizing flail chest. According to the author, this response is correct. More details and/or a list of references used by the author in preparing this simulation would be helpful to the user.

Drug Overdose. In this simulation, the patient arrives in the ER intoxicated and suspected of taking a drug overdose. Information is collected, and recommendations to collect additional diagnostic data or initiate therapy are required. Clinical deterioration occurs in the respiratory ICU, and ventilatory support is needed. The student must recommend ventilator setting changes based on blood gas results, weaning parameters, and hemodynamics. As an instructor in a respiratory care program, I prefer additional detail in answer section frames—this provides a study tool for students. For example, one answer response suggests, "P_{50} is an expensive test." There is no reference to support this statement. A list of references accompanying the simulation may help answer questions from users.

Neonate I. A high-risk mother gives birth to a premature newborn with Apgar scores of 4 and 3 at 1 and 5 minutes. Appropriate resuscitation efforts are needed, and recommendations to place the newborn on continuous positive airway pressure (CPAP), wean, extubate and place in an oxygen hood are required.

Pulmonary Rehabilitation. A 65-year-old man arrives at the ER with a history of increasing shortness of breath for 2 days. The simulation participant must evaluate this patient and recommend oxygen therapy, additional laboratory studies, and bronchial hygiene therapy. Assessing the patient's response to aerosol therapy and airway management, evaluating bedside spirometry, and recommending a discharge plan make this simulation unique to the set and certainly worthwhile to the student.

Muscular Dystrophy. A pediatric patient is admitted in respiratory distress for evaluation and recommendation of diagnostic tests, oxygen, and bronchial hygiene therapy. The patient develops increasing respiratory distress and requires additional therapy. This simulation includes IPPB, and the student must recommend the appropriate breathing technique for this treatment. I suggest that the author adds a breath-hold maneuver to the IPPB breathing instructions to improve distribution of ventilation. Currently, this detail is excluded. I appreciated the addition of a review of postural drainage positions for chest physical therapy and encourage more questions to test the student's knowledge of this topic. In the answer section, blood urea nitrogen (BUN) is explained as a test used to assess liver function; it is more correctly a test of renal function.

Neonate II. A baby is delivered by Cesarean section in a rural hospital and admitted to a larger, community medical center's neonatal intensive care unit for assessment and management. The baby develops respiratory difficulty requiring the student to make decisions about CPAP, oxygen therapy, the position of the endotracheal tube, the patient's readiness for extubation, and postextubation therapy. This simulation gives the student exposure to the type of neonatal patient who may be encountered on the Registry exam or in an actual clinical setting. The simulation ends abruptly when the newborn develops a pneumothorax. Management to a successful end unless an incorrect path had been chosen during the simulation with a pneumothorax resulting from poor decision-making might be preferable.

Epiglottitis. The student is asked to treat a pediatric patient with clinical signs of

*Hyperactive Software, 6709 128th Avenue SE, Bellview WA 98006, (206) 641-7534.

epiglottitis. Airway management is again emphasized, and the student may choose the treatment alternatives–oxygen therapy, aerosol therapy and ventilation techniques. A reference is made to a "grape juice" test which is described in the answer section as "A test for aspiration." Logical as it seems, there is no reference to direct students to this information.

Head Trauma. Students have an opportunity to evaluate intracranial pressures and to accomplish hyperventilation by mechanical ventilation in this motor vehicle accident patient. The patient develops a pulmonary infection during the course of mechanical ventilation that necessitates recommendations for additional laboratory studies and bronchial hygiene therapy.

Acute Respiratory Distress Syndrome (ARDS). This simulation provides a review of invasive hemodynamic values as the clinical picture of ARDS develops in this postoperative patient. A trial of CPAP is unsuccessful, and intubation with mechanical ventilation is required. Optimal positive end expiratory pressure and a review of static lung compliance are included. Although this simulation gives the student a taste for ventilator management, ie, changes to affect oxygenation, I favor a comprehensive review of ventilator management from start to finish within a simulation. The author's intent appears to focus on one particular ventilation/oxygenation problem per simulation.

Throughout the series, breath sounds are described as "rales" and "rhonchi" in comparison to the updated "crackles" and "wheezes." In most cases, the terms crackles and wheezes are added in parentheses after rales and rhonchi to further describe them and eliminate confusion for the user. Spelling and punctuation errors are infrequent. Student instructions to execute and move through the simulations are adequate although the Macintosh Hypercard version is considerably more user-friendly than the IBM. Immediate feedback is provided upon selection of responses and completion of the simulation, with alternatives provided for the user (ie, repeat the simulation or review all answers). Although the answer sections lack some detail, they may stimulate additional research and discussion among students as they search for a reference to defend their decisions and review the critical thinking that preceded them.

These clinical simulations are an affordable method for providing a variety of clinical situations to prepare students to apply classroom theory to the health-care setting, prepare for the national Registry exam, and enhance critical thinking. Because of the content and the comprehensiveness of the decisions required, I believe that the Rehab, COPD, Chest Trauma, Neonate II, Epiglottis simulations are the most valuable learning tools. Chest Trauma II and Drug Overdose are useful; however, I believe that the Cystic Fibrosis and Neonate I simulations are of limited value, even though they reinforce important basic skills.

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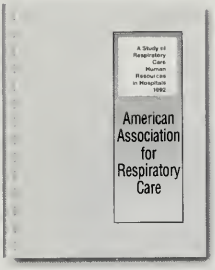
The AARC Human Resources Survey: A Study of Respiratory Care Human Resources in Hospitals



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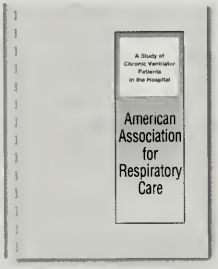

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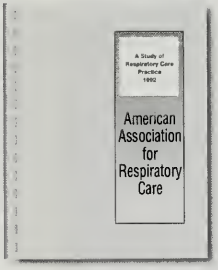

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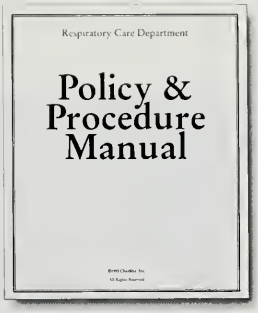

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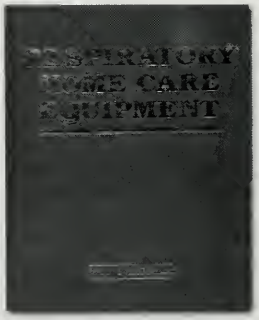

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TUBERCULOSIS CONTROL TRAINING. BioSafety Systems introduces the TB Exposure-Control Program with procedural guidelines and sample forms. According to BioSafety, the program is an excellent starting point for the creation of a TB Exposure Control Program. The program helps health-care professionals develop a program that incorporates CDC and OSHA guidelines. The program includes references, sample forms for documentation, and a 30-minute training video, as well as other materials from CDC. As a service, users are automatically informed of any important changes in regulations. For details, write to BioSafety Systems, Dept RC, 10225 Willow Creek Road, San Diego CA 92131, or call (800) 421-6556. Don't forget to mention RESPIRATORY CARE when you call.

REHABILITATION EQUIPMENT. Biodex Medical recently released the Upper Body Cycle for patients who have limited or no use of their lower extremities. Biodex claims that the Cycle helps geriatric or physically challenged patients regain or maintain vital cardiovascular health. The Cycle's design is especially useful for patients with limited range of motion and is adjustable for exercise above or below the shoulder, and while sitting, or standing. The retrocycling feature allows patients to pedal in reverse. In addition, the Cycle operates in 3 modes—isokinetic, constant power, and cardiac. Patient workloads may be selected for precision and resistance control. For information, mention RESPIRATORY CARE when you call (516) 924-9000 or write to Biodex Medical Systems, Dept RC, Brookhaven R & D Plaza, PO Box 702, Shirley NY 11967-0917.

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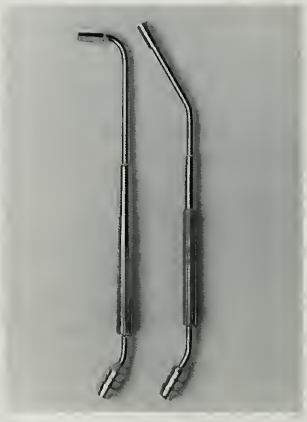
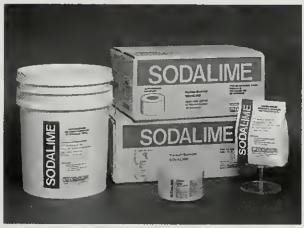


SUCTION PROBES. Pemco Medical now offers a full line of surgical stainless steel suction probes. Designed to reduce hazardous waste disposal costs, the probes may be sterilized for reuse, according to the manufacturer. In addition, the probes are designed to feel balanced in the hand. Two large-handle probes can accommodate four different interchangeable tips, and each probe handle has barbs for 1/4" and 3/8" ID tubes. Probe tips are available in a variety of sizes. For information, write Pemco Medical, Dept RC, 5663 Brecksville Road, Cleveland OH 44131. When calling (216) 524-2990, don't forget to mention RESPIRATORY CARE.

CLINICAL RESEARCH NETWORK. A clinical research network was recently developed for asthma and allergy researchers. According to Dr James Kemp—president of the network, the CRN/Asthma and Allergy Network Inc

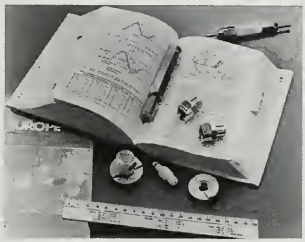
was developed to provide collective clinical research expertise to respiratory care researchers. The network includes 26 respiratory, allergy, and asthma research sites in 20 states and combines the expertise of more than 150 clinicians. The organization's objective is to offer the pharmaceutical, biotechnology, and medical device marketplace a time-efficient, cost-effective network to aid in research and development decisions, and to conduct clinical trials for new drug applications and for post-approval marketing support. Aligned with Respiratory Research Institute/LLC, the network offers consultation agreements for a variety of allergy and asthma research processes. For information, call (302) 791-7771 or write to CRN/Allergy & Asthma Network Inc, Dept RC, Silverside Carr Executive Center, 501 Silverside Road, Suite 31, Wilmington DE 19809. Please mention RESPIRATORY CARE when calling.

TRACHEOSTOMY-TUBE STABILIZER. Zipper Medical Inc now offers a tracheostomy tube neck band created specifically to stabilize the tube and prevent skin irritation and chaffing. According to Zipper Medical, the one-piece, hypoallergenic cotton pile with quick-release fasteners adjusts to fit a wide range of neck sizes. Available in adult and pediatric sizes, the neck band fits securely and comfortably. For information write to Zipper Medical Inc, Dept RC, PO Box 545 Kensington MD 20895 or call (800) 647-0172. Please mention RESPIRATORY CARE when you call.



MANUFACTURER ISO CERTIFIED. SODALIME carbon dioxide absorbent, distributed by Puritan-Bennett Corporation, has been awarded ISO 9002 Certification. Puritan-Bennett claims it is the first carbon dioxide absorbent manufacturer in North America to receive this quality certification. SODALIME brand of Soda Lime USP/NF carbon dioxide absorbent is widely used in hospitals and surgery centers to remove carbon dioxide from anesthesia breathing circuits, making it possible for anesthetic gases to be rebreathed by the patient. Call for more information and mention RESPIRATORY CARE when you call (913) 752-6722.

NEONATAL INFRARED THERMOMETER. Exergen Corporation announces the LighTouch Neonate Axillary Infrared Thermometer. This technological breakthrough incorporates the accuracy, speed and safety of infrared technology with the clinically proven reliability of axillary site temperature. According to the manufacturer, the thermometer can measure neonate temperatures in less than a second with no tissue cool down, can be used on wet skin, in incubators, during phototherapy and in the presence of radiant heaters, warming pads, or other heat sources. The thermometer may also be used to instantly verify ambient, incubator, or medication temperatures. The device can withstand demanding clinical use, including dropping, and may be disinfected with bleach or by gas sterilization. One model may be fully enclosed in a disposable protective sheath; the other model uses disposable probe covers. Both models come with a lifetime warranty. For details, write to Exergen Corporation, Dept RC, 1 Bridge Street, Newton MA 02158, or call (800) 422-3006. Remember to mention RESPIRATORY CARE when you call.



METRIC-SIZE COUPLINGS. Colder Products Company offers coupling products in new metric sizes. According to the manufacturer, the new CPC couplings have built-in ferruleless fittings for 4-, 8-, and 10-mm OD tubing, and hose barbs to 19-mm-ID flexible tubing. Ferruleless fittings designed for semirigid tubing such as polyethylene, nylon, and polyurethane and new pipe-thread sizes are also ready-to-buy. In addition, panel-mound, in-line, and pipe-thread configurations are available with or without shutoff valves. For more information about metric couplings, write to Colder Products Company, Dept RC, 1001 Westgate Drive, St Paul MN 55114 or call (612) 645-0091. When you call, please mention RESPIRATORY CARE.

IISPs...

Individual Independent Study Packages Assist Practitioners In Expanding Their Respiratory Care Knowledge

Smoking Cessation

Nicotine Dependency Evaluation and Treatment
Helps you understand the physiologic effects of nicotine and the tests and questions used to evaluate dependency. Provides an understanding of nicotine, its effects on brain chemistry, its measurement in bodily fluids, and the value of self-tests to determine addiction level. Also teaches about nicotine replacement and how to enhance it with behavioral counseling.
Item SC1 $10 each ($15 nonmembers)

Bedside Counseling of the Hospitalized Smoker
Prepares you for the role of a smoking cessation counselor to hospital inpatients by teaching assessment and bedside counseling. Help patients cope with nicotine withdrawal, increase motivation for permanent cessation, and understand the factors contributing to relapse.
Item SC2 $10 each ($15 nonmembers)

Neonatal

Recognition and Stabilization of the Premature Infant in Respiratory Distress
This IISP helps you identify primary risk factors associated with premature birth. Discusses and explains the importance of providing a neutral-thermal environment for the premature infant and the necessity of continuous noninvasive oxygen monitoring of the premature infant on supplemental oxygen. You will also learn to recognize aberrant blood gases in the neonate and oxygen therapy initiation with an oxygen hood.
Item NN1 $10 each ($15 nonmembers)

Clinical Science

Transpulmonary Pressure Changes in Breathing
Discusses the pulmonary pressures and how they vary during normal breathing and in special situations. Instructs on barometric, alveolar, intrapleural, transpulmonary, chest wall, and transthoracic pulmonary pressures.
Item CS5 $10 each ($15 nonmembers)

Lung Mechanics
Assists you in understanding the pulmonary characteristics of compliance and resistance, how these characteristics can change, and their influence on mechanical ventilator performance.
Item CS6 $10 each ($15 nonmembers)

Practical Application of Gas Laws
A common-sense approach to the gas laws and how to apply them to respiratory care. Defines the laws of Boyle, Charles, Gay-Lussac, Dalton, Graham, and Henry.
Item CS7 $10 each ($15 nonmembers)

Tonicity of Solutions and the Respiratory Tract
Gives you an understanding of how to predict the effects of therapeutic solutions on the respiratory tract and how to utilize them in the treatment of various respiratory tract pathologies.
Item CS8 $7 each ($12 nonmembers)

Carbon Monoxide Inhalation: Introduction to Physiologic Effects and Respiratory Management
Explains the effects of carbon monoxide inhalation on oxygen transport mechanisms in the body and the use of CO in physiologic tests of pulmonary function. Also covers recognition signs and symptoms, treatment of CO poisoning, and the equipment used to administer therapy.
Item CS9 $10 each ($15 nonmembers)

Electrical Safety in Respiratory Therapy I: Basic Electrical Circuitry
Enables you to relate the basic principles of electrical theory to commonly used equipment and procedures in the clinical environment and provides you with an understanding of electrical safety in respiratory care.
Item CS12 $7 each ($12 nonmembers)

Electrical Safety in Respiratory Therapy II: Identification of Electrical Hazards
Helps you identify and, where possible, minimize or eliminate common electrical hazards. Teaches differentiation between electrical hazards and which patients and hospital personnel are exposed.
Item CS13 $7 each ($12 nonmembers)

Bronchodilators I: Sympathomimetic Amines
Understand the results of stimulating the autonomic nervous system and the use of sympathomimetic drugs to accomplish bronchodilation. Exposes you to basic aspects of adrenergic bronchodilators and the patient situations for which they are indicated.
Item CS14 $10 each ($15 nonmembers)

Bronchodilators II: Anti-Cholinergics and Xanthines
Identifies the three categories of drugs that promote bronchodilation and the mechanism of action for each. This package also provides examples of drugs in each category. Clinical situations are presented with methods of bronchodilaton and rationale for method selected.
Item CS15 $10 each ($15 nonmembers)

Calculation and Preparation of Respiratory Medications
Teaches you how to calculate and prepare percentage solutions, ration solutions, solutions prepared from solutes less than 100 percent in strength, and how to prepare dosages from powdered drugs.
Item CS16 $10 each ($15 nonmembers)

Microbiology for Respiratory Therapy: A Review of Microbial Growth and Cross-Contamination
Provides an overview of microbiology in respiratory care, including groups and characteristics of microbes, requirements for microbial growth, cross-contamination, and prevention of transmission.
Item CS17 $9 each ($14 nonmembers)

Classification of Mechanical Ventilators I
Outlines the basic concepts of ventilator classification and a mathematical model of the respiratory system that provides the basis for classifying ventilator control systems. Also teaches the specific criteria for determining whether a ventilator primarily controls pressure, volume, or flow.
Item CS18 $10 each ($15 nonmembers)

Classification of Mechanical Ventilators II
You will learn detailed information about the control of ventilation in terms of switching from inspiration to expiration. You will also be introduced to the common drive mechanisms used in various mechanical devices.
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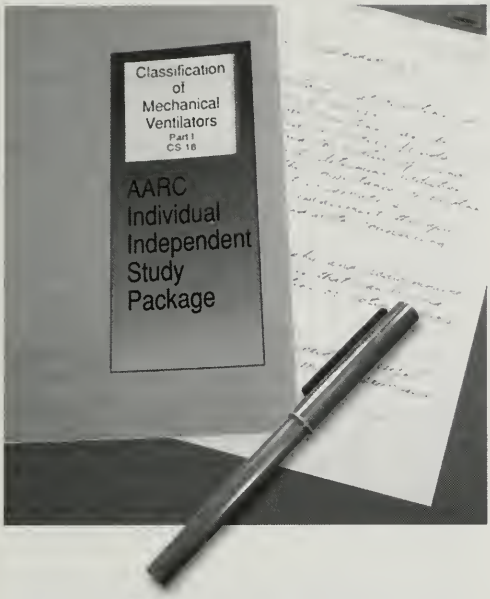

Classification of Mechanical Ventilators III
Explores the output waveforms that ventilators produce and their clinical implications. Instructs in the various modes of ventilation in terms of the specific combinations of control characteristics along with ventilator performance testing and the practical application of ventilator classification.
Item CS20 $10 each
($15 nonmembers)

Ventilation/Perfusion Relationships in Health and Disease
Covers the different degrees of hypoxia commonly encountered in patients with pulmonary disease. Focuses on ventilation/perfusion abnormalities, which account for much of the severe hypoxia in COPD, with alveolar hypoventilation also a contributing factor.
Item CS21 $10 each
($15 nonmembers)

Patient Evaluation

Pulmonary Function Assessment I: Basic Screening Studies
Understand simple spirometry results, the definition of obstructive and restrictive pulmonary disorders, and the basic pathology involved in each. Covers the four reasons for simple spirometry tests, the common parameters of lung function, and the results that are indicative of obstructive and restrictive pulmonary disorders.
Item PE3 $8 each
($13 nonmembers)

Pulmonary Function Assessment II: Bedside Studies
Familiarizes you with the use and interpretation of bedside pulmonary function tests. Learn how pulmonary function test results are used to assess ventilatory ability and which tests are commonly used. Also learn the measuring instrument required for each test, how it is performed, and what is significant about the results.
Item PE4 $7 each
($12 nonmembers)

Pulmonary Function Assessment III: Lung Volume Determination and Closing Volume Studies
Assists in the recall of the eight subdivisions of total lung volume and their individual characteristics in health and disease. Discusses the measurement of lung volume by nitrogen washout, helium dilution, and closing volume techniques.
Item PE5 $7 each
($12 nonmembers)

Body Plethysmography
Explains the theory of body plethysmography and its use in the measurement of FRC and airway resistance. Defines and describes Boyle's Law, thoracic volume, airway resistance and its normal values, and the measurement of airway resistance.
Item PE6 $9 each
($14 nonmembers)

Sources of Error in the Determination of Blood Gas Values and pH
Familiarizes you with the errors that may occur in the analysis of blood gases and pH. These are often the most important laboratory data used in the diagnosis and treatment of pulmonary disease, and errors in these values can result in deleterious effects on patient care.
Item PE7 $10 each
($15 nonmembers)

Temperature Adjustment of Blood Gases and pH
Teaches you the effects of abnormal body temperature on blood gas and pH values. Blood gas values and pH are determined at 37 degrees Celsius, and this package teaches you how to adjust these values.
Item PE8 $10 each
($15 nonmembers)

Updated! Plotting Static and Dynamic Compliance Curves During Mechanical Ventilation
Learn how to compute and record static compliance curves and dynamic characteristics. Also learn the procedure of obtaining pressure-volume measurements and interpret the compliance characteristics measurements.
Item PE9 $10 each
($15 nonmembers)

Arterial Blood Gas Interpretation
Teaches you ABG interpretation in order that the prescribed therapy can be administered in a knowledgeable manner. Describes a systematic method that allows you to correctly classify the acid/base dysfunction and to relate the diagnosis concisely and coherently.
Item PE10 $10 each
($15 nonmembers)

Clinical Practice

Chest Tubes and Pleural Drainage
Helps you understand the purpose of pleural drainage, how it might affect the patient's respiratory status, and what precautions you must take when working with patients who are receiving pleural drainage.
Item CP3 $10 each
($15 nonmembers)

Tracheal Intubation I: Upper Airway Anatomy and Goals of Intubation
After completing this IISP, you will understand the rationale for tracheal intubation and be able to identify the important landmarks of upper airway anatomy.
Item CP4 $7 each
($12 nonmembers)

Tracheal Intubation II: Routes of Intubation
Describes the four routes of tracheal intubation and some advantages and hazards of each. Also presents the process for selecting the most suitable route in a given situation.
Item CP5 $7 each
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Tracheal Intubation III: Equipment Procedures for Intubation
Covers the selection of the proper equipment necessary to perform endotracheal intubation and to ensure that they are in working order.
Item CP6 $8 each
($13 nonmembers)

Respiratory Management of Neuromuscular Crisis
Teaches respiratory management of patients with ventilatory failure caused by a neuromuscular disorder. Provides a basic understanding of how neuromuscular conditions lead to ventilatory insufficiency and the considerations to be taken when working with these patients.
Item CP7 $7 each
($12 nonmembers)

Respiratory Management of Flail Chest
This study package helps you increase your understanding of the pathophysiology of flail chest and the respiratory management of patients who have sustained chest wall trauma that results in a flail chest.
Item CP8 $10 each
($15 nonmembers)

Respiratory Management of Head Trauma
Teaches identification of the five physical signs indicative of head trauma and explains the development of respiratory failure secondary to trauma. Also discusses airway management, drug therapy, ventilator parameters, acid-base status, and measures to be taken to maintain the appropriate pH, P_{aCO_2} and P_{aO_2}.
Item CP9 $10 each
($15 nonmembers)

RESPIRATORY CARE

Manuscript Preparation Guide

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Research Article: A report of an original investigation (a study). It includes a Title Page, Abstract, Introduction, Methods, Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Evaluation of Device/Method/Technique: A description and evaluation of an old or new device, method, technique, or modification. It has a Title Page, Abstract, Introduction, Description of Device/Method/Technique, Evaluation Methods, Evaluation Results, Discussion, Conclusions, Product Sources, Acknowledgments, References, Tables, Appendices, Figures, and Figure Captions.

Case Report: A report of a clinical case that is uncommon, or was managed in a new way, or is exceptionally instructive. All authors must be associated with the case. A case-managing physician must either be an author or furnish a letter approving the manuscript. Its components are Title Page, Abstract, Introduction, Case Summary, Discussion, References, Tables, Figures, and Figure Captions.

Review Article: A comprehensive, critical review of the literature and state-of-the-art summary of a pertinent topic that has been the subject of at least 40 published research articles. Title Page, Outline, Introduction, Review of the Literature, Summary, Acknowledgments, References, Tables, Appendices, and Figures and Captions may be included.

Overview: A critical review of a pertinent topic that has fewer than 40 published research articles.

Update: A report of subsequent developments in a topic that has been critically reviewed in this Journal or elsewhere.

Point-of-View Paper: A paper expressing personal but substantiated opinions on a pertinent topic. Title Page, Text, References, Tables, and Illustrations may be included.

Special Article: A pertinent paper not fitting one of the foregoing categories may be acceptable as a Special Article. Consult with the Editor before writing or submitting such a paper.

Editorial: A paper drawing attention to a pertinent concern; it may present an opposing opinion, clarify a position, or bring a problem into focus.

Letter: A signed communication about prior publications in this Journal or about other pertinent topics. Tables and illustrations may be included. Mark "For publication."

Blood Gas Corner: A brief, instructive case report involving blood-gas values—with Questions, Answers, and Discussion.

Drug Capsule: A mini-review paper about a drug or class of drugs that includes discussions of pharmacology, pharmacokinetics, and pharmacotherapy.

Kittredge's Corner: A brief description of the operation of respiratory care equipment—with information from manufacturers and editorial comments and suggestions.

PFT Corner: Like Blood Gas Corner, but involving pulmonary function tests.

Test Your Radiologic Skill: Like Blood Gas Corner, but involving pulmonary medicine radiography and including one or more radiographs, may involve imaging techniques other than conventional chest radiography.

Review of Book, Film, Tape, or Software: A balanced, critical review of a recent release.

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tification anywhere except on the title page. Repeat title only (no authors) on the abstract page. Begin each of the following on a new page: Title Page, Abstract, Text, Product-Sources List, Acknowledgments, References, each Table, and each Appendix. Use standard English in the first person and active voice.

Center main section headings on the page and type them in capital and small letters (eg, Introduction, Methods, Results, Discussion). Begin subheadings at the left margin and type them in capital and small letters (eg, Patients, Equipment, Statistical Analysis).

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Article in a journal carrying pagination throughout volume:

1. Rau JL, Harwood RJ. Comparison of nebulizer delivery methods through a neonatal endotracheal tube: a bench study. Respir Care 1992;37(11):1233-1240.

Article in a publication that numbers each issue beginning with Page 1:

2. Bunch D. Establishing a national database for home care. AARC Times 1991;15(Mar):61,62,64.

Corporate author journal article:

3. American Association for Respiratory Care. Criteria for establishing units for chronic ventilator-dependent patients in hospitals. Respir Care 1988;33(11):1044-1046.

Article in journal supplement: (Journals differ in their methods of numbering and identifying supplements. Supply sufficient information to promote retrieval.)

4. Reynolds HY. Idiopathic interstitial pulmonary fibrosis. Chest 1986;89(3, Suppl):139S-143S.

Abstract in journal: (Abstracts citations are to be avoided. Those more than 3 years old should not be cited.)

5. Stevens DP. Scavenging ribavirin from an oxygen hood to reduce environmental exposure (abstract). Respir Care 1990;35(11): 1087-1088.

Editorial in journal:

6. Enright P. Can we relax during spirometry? (editorial). Am Rev Respir Dis 1993;148(2):274.

Editorial with no author given:

7. Negative-pressure ventilation for chronic obstructive pulmonary disease (editorial). Lancet 1992;340(8833):1440-1441.

Letter in journal:

8. Aelony Y. Ethnic norms for pulmonary function tests (letter). Chest 1991;99(4):1051.

Paper accepted but not yet published:

9. Hess D. New therapies for asthma. Respir Care (year, in press).

Personal author book: (For any book, specific pages should be cited whenever possible.)

10. DeRemee RA. Clinical profiles of diffuse interstitial pulmonary disease. New York: Futura, 1990:76-85.

Corporate author book:

11. American Medical Association Department of Drugs. AMA drug evaluations, 3rd ed. Littleton CO: Publishing Sciences Group, 1977.

Chapter in book with editor(s):

12. Pierce AK. Acute respiratory failure. In: Guenter CA, Welch MH, editors. Pulmonary medicine. Philadelphia: JB Lippincott, 1977.

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	Examination Date	Application Deadline
CRTT:	July 15, 1995	May 1, 1995
	November 11, 1995	September 1, 1995
RRT:	June 3, 1995	February 1, 1995
	December 2, 1995	August 1, 1995
CPFT:	June 3, 1995	April 1, 1995
RPFT:	December 2, 1995	September 1, 1995

Fee Schedule

CRTT	
—new applicant:	$ 90.00
—reapplicant:	$ 60.00
RRT Written and Clinical Simulation	
— new applicant.	$ 190.00
—reapplicant:	$ 160.00
Written Registry Only	
—new applicant:	$ 90.00
—reapplicant.	$ 60.00
Clinical Simulation Only—new & reapplicant:	$ 100.00
CPFT	
—new applicant:	$ 100.00
—reapplicant:	$ 80.00
RPFT	
—new applicant:	$ 150.00
—reapplicant:	$ 130.00
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—new applicant:	$ 150.00
—reapplicant:	$ 130.00

	Active	Inactive
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RRT Recredentialing:		
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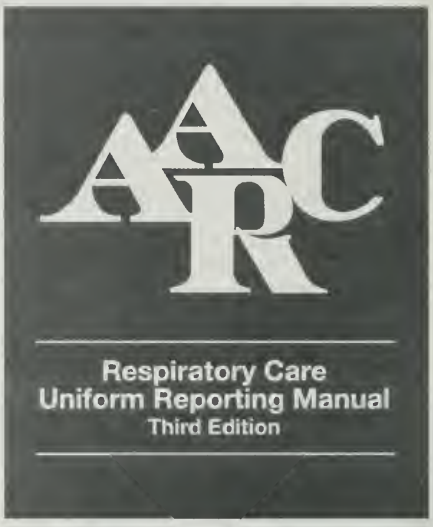

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AARC & AFFILIATES

July 14–16 in Vail, Colorado. The AARC presents the Summer Forum, with emphasis on management and education topics. Consult the April issue of AARC Times for program details and registration information.

August 23–25 in Albuquerque, New Mexico. The NMSRC announces its Annual Summer Convention at the Albuquerque Convention Center. The program features an I.V. class and a bronchoalveolar lavage class; other topics include postacute care and pressure-control ventilation. Contact Brooke Patterson of THC Hospital at (505) 242-4444.

September 21–22 in Napa, California. Chapter 10 of the CSRC, the American Lung Association of the Redwood Empire and the respiratory therapy program at Napa Valley College present the 13th Annual Napa Valley Conference. Topics include therapist-driven protocols, primary care concerns in HIV, sleep-related breathing disorders, pediatric trauma triage, and ethics in ventilator termination. Contact Kate Benscoter at (707) 253-3145.

September 21–22 in Pittsburgh, Pennsylvania. The PSRC announces the 22nd Annual Pulmonary Physiology and Medicine Conference at the Sheraton Hotel. Events include general and workshop sessions as well as a physicians forum and a continuum of care tract. Contact Tracy Painter at (412) 622-4100 or Joe Garuccio at (412) 359-3031.

September 26–27 in Honolulu, Hawaii. The HSRC and the American Lung Association of Hawaii host the 22nd Annual Respiratory Care Conference at the Hilton Hawaiian Village Hotel. Topics include treatment of airway obstruction by therapist-driven protocol, clinical trials in liquid ventilation, an update on high-frequency ventilation, newer forms of mechanical ventilation by protocol, and metabolic monitoring. Contact Wendell Inouye RRT, at Kapiolani Medical Center for Women and Children, (808) 973-8689, fax (808) 973-4930.

OTHER MEETINGS

August 25–27 in Islamorado, Florida. Sunset Seminars/ Robbins & Associates presents their Annual Bikini Beach Retreat at the Holiday Isle Resort and Marina, with 8 continuing education hours on "Respiratory Care in the Rehabilitation Setting." Contact Sunset Seminars/ Robbins & Associates, 19800 SW 180 Ave, Suite 127, Miami FL 33187, (305) 232-1908.

September 22-23, in Nagoya, Japan. The Asia Pacific Association for Respiratory Care (APARC) presents its Fourth Congress. A stellar international faculty addresses new modes of treatment and mechanical ventilation and the latest in patient management. For registration information and travel arrangements, contact Travel Plaza International Chubu Inc, Ishida Bldg 9F, 4-25-21 Meieki, Nakamura-ku, Nagoya 450, Japan. Phone: +81-52-561-9880/8655; Fax: +81-52-541-2520.

September 28–October 1 in Dearborn, Michigan. The Extracorporeal Life Support Organization (ELSO) hosts its 7th Annual ELSO Meeting at the Ritz Carlton Hotel. The deadline for abstract submission is July 15. Call (313) 998-6600.

October 6 in New York, New York. The Lenox Hill Hospital Department of Medicine's Pulmonary Section presents a symposium entitled "Pulmonary Medicine: Looking at the Future." Symposium topics include clinical and investigative forefronts. Contact Pulmonary Section, Department of Medicine, Lenox Hill Hospital, 100 E 77th St, New York NY 10021, (212) 434-2145.

October 12–15 in Minneapolis, Minnesota. The American Association of Cardiovascular and Pulmonary Rehabilitation hosts its 10th Annual Meeting, "Building on Success—A Decade of Progress," at the Minneapolis Convention Center. For registration and hotel information, contact Michele Johnson (608) 831-6989.

October 29–November 2 in New York, New York. The 61st Annual International Scientific Assembly sponsored by the American College of Chest Physicians (ACCP) at the New York Hilton and Towers is entitled "Chest 1995: Prevention and Diagnosis of Chest Disease." Contact the ACCP, 3300 Dundee Rd, Northbrook IL 60062-2348.

November 9–10 in Lyon, France. The Association for Diagnosis and Treatment of Sleep Apnea Syndrome announces an international conference entitled "From Snoring to Sleep Apnea Syndrome: Therapeutic Approach." The conference looks at therapeutic options and their practical management regarding the treatment of an abnormal increase of upper airway resistance during sleep. The meeting offers plenary sessions, poster sessions, a technical exhibit, and clinical and practical workshops. Contact B Hautier or S Sazio, Hôpital de la Croix Rousse, SRMAR, 93 Grande Rue de la Croix Rousse, 69317 LYON Cedex 04, France, or call (33) 78 39 08 43 or fax (33) 78 39 58 63.

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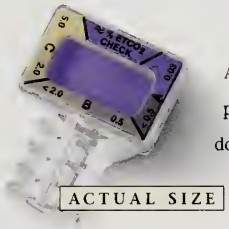

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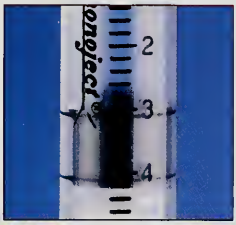

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